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NEWS 1		Web Page for STN Seminar Schedule - N. America
NEWS 2	AUG 06	CAS REGISTRY enhanced with new experimental property tags
NEWS 3	AUG 06	FSTA enhanced with new thesaurus edition
NEWS 4	AUG 13	CA/CAplus enhanced with additional kind codes for granted patents
NEWS 5	AUG 20	CA/CAplus enhanced with CAS indexing in pre-1907 records
NEWS 6	AUG 27	Full-text patent databases enhanced with predefined patent family display formats from INPADOCDB
NEWS 7	AUG 27	USPATOLD now available on STN
NEWS 8	AUG 28	CAS REGISTRY enhanced with additional experimental spectral property data
NEWS 9	SEP 07	STN AnaVist, Version 2.0, now available with Derwent World Patents Index
NEWS 10	SEP 13	FORIS renamed to SOFIS
NEWS 11	SEP 13	INPADOCDB enhanced with monthly SDI frequency
NEWS 12	SEP 17	CA/CAplus enhanced with printed CA page images from 1967-1998
NEWS 13	SEP 17	CAplus coverage extended to include traditional medicine patents
NEWS 14	SEP 24	EMBASE, EMBAL, and LEMBASE reloaded with enhancements
NEWS 15	OCT 02	CA/CAplus enhanced with pre-1907 records from Chemisches Zentralblatt
NEWS 16	OCT 19	BEILSTEIN updated with new compounds
NEWS 17	NOV 15	Derwent Indian patent publication number format enhanced
NEWS 18	NOV 19	WPIX enhanced with XML display format
NEWS 19	NOV 30	ICSD reloaded with enhancements
NEWS 20	DEC 04	LINPADOCDB now available on STN
NEWS 21	DEC 14	BEILSTEIN pricing structure to change
NEWS 22	DEC 17	USPATOLD added to additional database clusters
NEWS 23	DEC 17	IMSDRUGCONF removed from database clusters and STN
NEWS 24	DEC 17	DGENE now includes more than 10 million sequences
NEWS 25	DEC 17	TOXCENTER enhanced with 2008 MeSH vocabulary in MEDLINE segment
NEWS 26	DEC 17	MEDLINE and LMEDLINE updated with 2008 MeSH vocabulary
NEWS 27	DEC 17	CA/CAplus enhanced with new custom IPC display formats
NEWS 28	DEC 17	STN Viewer enhanced with full-text patent content from USPATOLD
NEWS 29	JAN 02	STN pricing information for 2008 now available
NEWS 30	JAN 16	CAS patent coverage enhanced to include exemplified prophetic substances
NEWS 31	JAN 28	USPATFULL, USPAT2, and USPATOLD enhanced with new custom IPC display formats
NEWS 32	JAN 28	MARPAT searching enhanced

Updated Search

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NEWS 33 JAN 28 USGENE now provides USPTO sequence data within 3 days of publication
NEWS 34 JAN 28 TOXCENTER enhanced with reloaded MEDLINE segment
NEWS 35 JAN 28 MEDLINE and LMEDLINE reloaded with enhancements

NEWS EXPRESS 19 SEPTEMBER 2007: CURRENT WINDOWS VERSION IS V8.2, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 19 SEPTEMBER 2007.

NEWS HOURS STN Operating Hours Plus Help Desk Availability
NEWS LOGIN Welcome Banner and News Items
NEWS IPC8 For general information regarding STN implementation of IPC 8

Enter NEWS followed by the item number or name to see news on that specific topic.

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FILE 'HOME' ENTERED AT 19:45:51 ON 30 JAN 2008

=> file reg

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.42	0.42

FILE 'REGISTRY' ENTERED AT 19:47:05 ON 30 JAN 2008
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Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 29 JAN 2008 HIGHEST RN 1001040-86-3
DICTIONARY FILE UPDATES: 29 JAN 2008 HIGHEST RN 1001040-86-3

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH June 29, 2007

Please note that search-term pricing does apply when conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

http://www.cas.org/support/stngen/stndoc/properties.html

=>
Uploading C:\Documents and Settings\brobinson1\My Documents\stnweb\Queries\bnvbtgy.str

Updated Search

09890219

```
L1      STRUCTURE UPLOADED

=> d l1
L1 HAS NO ANSWERS
L1              STR
*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

Structure attributes must be viewed using STN Express query preparation.


=> s l1
SAMPLE SEARCH INITIATED 19:53:52 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED -    66196 TO ITERATE

  3.0% PROCESSED     2000 ITERATIONS                                50 ANSWERS
INCOMPLETE SEARCH (SYSTEM LIMIT EXCEEDED)
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS:  ONLINE  **INCOMPLETE**
                        BATCH   **COMPLETE**
PROJECTED ITERATIONS:      1308593 TO  1339247
PROJECTED ANSWERS:           57591 TO    64209

L2           50 SEA SSS SAM L1

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Uploading C:\Documents and Settings\brobinson1\My
Documents\stnweb\Queries\anfjut,str.str

L3      STRUCTURE UPLOADED

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SAMPLE SCREEN SEARCH COMPLETED -    45493 TO ITERATE

  4.4% PROCESSED     2000 ITERATIONS                                50 ANSWERS
INCOMPLETE SEARCH (SYSTEM LIMIT EXCEEDED)
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS:  ONLINE  **COMPLETE**
                        BATCH   **COMPLETE**
PROJECTED ITERATIONS:       897125 TO   922595
PROJECTED ANSWERS:           55436 TO    61934

L4           50 SEA SSS SAM L3

=> s l3 full
THE ESTIMATED SEARCH COST FOR FILE 'REGISTRY' IS 177.90 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N or END:y
FULL SEARCH INITIATED 19:54:46 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED -   909835 TO ITERATE

 95.5% PROCESSED   868916 ITERATIONS                           54573 ANSWERS

 98.7% PROCESSED   898118 ITERATIONS                           57549 ANSWERS

100.0% PROCESSED   909835 ITERATIONS                           58230 ANSWERS
SEARCH TIME: 00.00.36
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Updated Search

09890219

L5 58230 SEA SSS FUL L3

=> file hcaplus
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 184.34 184.76

FILE 'HCAPLUS' ENTERED AT 19:55:28 ON 30 JAN 2008
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

FILE COVERS 1907 - 30 Jan 2008 VOL 148 ISS 5
FILE LAST UPDATED: 29 Jan 2008 (20080129/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s l5
L6 21413 L5

=> s l6 and matsuoka, h?/au
2662 MATSUOKA, H?/AU
L7 15 L6 AND MATSUOKA, H?/AU

=> d l7, ibib abs fhitstr, 1-15

L7 ANSWER 1 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2002:157810 HCAPLUS
DOCUMENT NUMBER: 136:217049
TITLE: Preparation of cyclic peptide derivatives as motilin receptor antagonists
INVENTOR(S): Matsuoka, Hiroharu; Sato, Tsutomu
PATENT ASSIGNEE(S): Chugai Seiyaku Kabushiki Kaisha, Japan
SOURCE: PCT Int. Appl., 89 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002016404	A1	20020228	WO 2001-JP7213	20010823

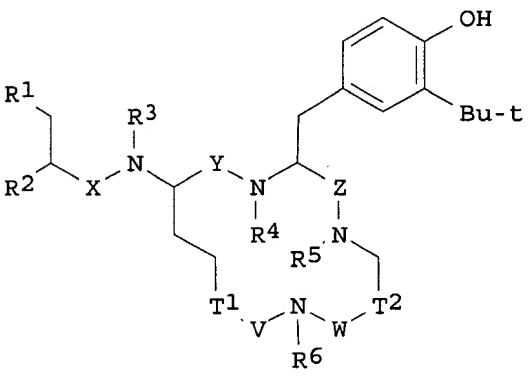
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,

Updated Search

CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2001080120	A5	20020304	AU 2001-80120	20010823
EP 1312612	A1	20030521	EP 2001-958426	20010823
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2003191053	A1	20031009	US 2003-362574	20030224
US 7018981	B2	20060328		
PRIORITY APPLN. INFO.:			JP 2000-253950 A	20000824
			WO 2001-JP7213 W	20010823

OTHER SOURCE(S): MARPAT 136:217049
GI



I

AB The title compds. I [T1 = (CH2)m; T2 = (CH2)n; R1 represents optionally substituted Ph, etc.; R2 represents amino, etc.; R3 to R6 each represents hydrogen, Me, etc.; V, W, X,Y, Z represent carbonyl or methylene; m is an integer of 0 to 2; and n is an integer of 0 to 3] are prepared In an in vitro test for motilin receptor antagonism, (2S-(2S,12S))-2-amino-N-(2-(3-tert-butyl-4-hydroxylphenylmethyl)-1,4,8-triaza-3,7,13-trioxocyclotridecan-12-yl)-3-(4-fluorophenyl)-N-methylpropionamide showed IC50 of 0.52 nM.

IT 401896-13-7P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation of cyclic peptide derivs. as motilin receptor antagonists)

RN 401896-13-7 HCAPLUS

CN β-Alanine, N-[(1,1-dimethylethoxy)carbonyl]-4-fluoro-L-phenylalanyl-N2-methyl-N6-[(phenylmethoxy)carbonyl]-L-lysyl-3-(1,1-dimethylethyl)-L-tyrosyl-, phenylmethyl ester (9CI) (CA INDEX NAME)

Absolute stereochemistry.

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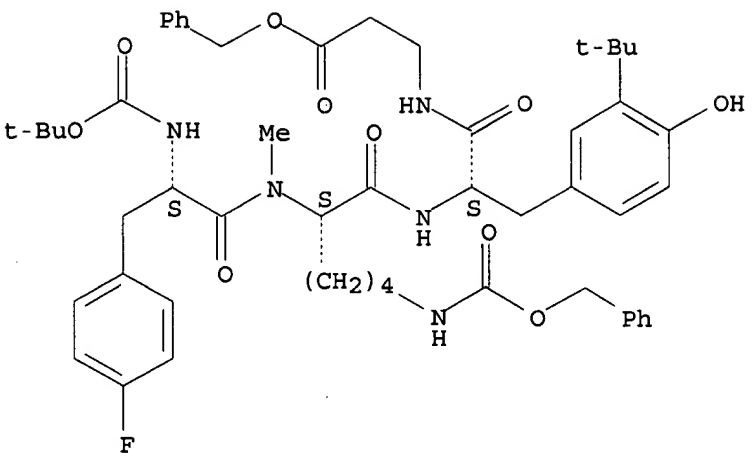


REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 2 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2000:535162 HCAPLUS
DOCUMENT NUMBER: 133:150920
TITLE: Preparation of peptides or analogs containing substituted phenethylamine moiety as motilin receptor antagonists
INVENTOR(S): Matsuoka, Hiroharu; Sato, Tsutomu; Takahashi, Tadakatsu; Kim, Dong Ick; Jung, Kyung Yun; Park, Chan Hee
PATENT ASSIGNEE(S): Chugai Seiyaku Kabushiki Kaisha, Japan
SOURCE: PCT Int. Appl., 403 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000044770	A1	20000803	WO 2000-JP444	20000128
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2359030	A1	20000803	CA 2000-2359030	20000128
EP 1149843	A1	20011031	EP 2000-901956	20000128
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
HU 2001005204	A2	20020429	HU 2001-5204	20000128
HU 2001005204	A3	20020528		
JP 3715202	B2	20051109	JP 2000-596026	20000128
NO 2001003684	A	20010928	NO 2001-3684	20010726
PRIORITY APPLN. INFO.:			JP 1999-20523 A	19990128
			JP 1999-283163 A	19991004

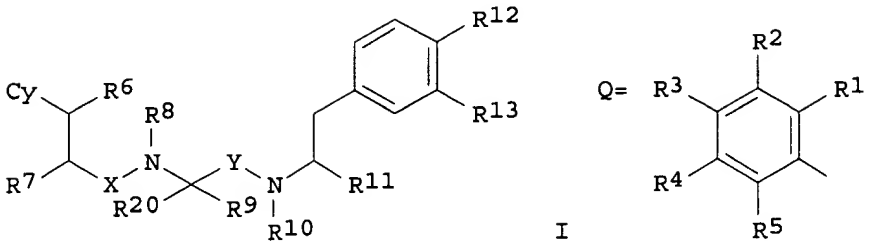
Updated Search

09890219

WO 2000-JP444 W 20000128

OTHER SOURCE(S): MARPAT 133:150920

GI



I

AB Substituted phenethylamine derivs. represented by general formula (I), hydrates of the same, or pharmaceutically acceptable salts thereof [wherein Cy is a group represented by general formula Q, an optionally substituted heterocyclic group, C3-7 cycloalkyl, or phenyl; R1, R1, R1, R1 and R5 are each hydrogen, halogeno, hydroxyl, amino, trifluoromethyl or cyano, at least one of R1-R5 being halogeno, trifluoromethyl or cyano; R6 represents hydrogen, (un)substituted linear or branched C1-3 alkyl, amino, or hydroxy; R8 represents hydrogen, Me, or ethyl; R9 represents (un)substituted linear or branched C1-6 alkyl, C2-6 alkenyl, or C2-6 alkynyl, C3-7 cycloalkyl, or (un)substituted Ph; R20 represents hydrogen, or (un)substituted linear or branched C1-3 alkyl or R9 and R20 together forms C3-7 cycloalkyl; R10 represents hydrogen, (un)substituted linear or branched C1-3 alkyl; R11 represents hydrogen or (un)substituted linear or branched C1-3 alkyl, (un)substituted carbamoyl, or carboxy; R12 represents hydroxy or linear or branched C1-4 alkoxy; R13 represents hydrogen, (un)substituted linear or branched C1-6 alkyl, C2-6 alkenyl, or alkynyl, etc.; X, Y represents carbonyl or CH2; provisos are given.], which exhibit motilin receptor antagonism and being useful as drugs for preventing digestive tract movement or high level of blood motilin. Thus, 3-methyl-2-methylaminobutyric acid 2-(3-tert-butyl-4-hydroxyphenyl)-1-(2-pyridylcarbamoyl)ethylamide (preparation given) was condensed with Boc-Phe(4-F)-OH using CMPI in the presence of Et3N in THF under ice-cooling for 4 h followed by treatment of the product with CF3CO2H in CH2Cl2 gave 2-((2-amino-3-(4-fluorophenyl)propanoyl)-N-methylamino)-3-methylbutyric acid 2-(3-tert-butyl-4-hydroxyphenyl)-1-(2-pyridylcarbamoyl)ethylamide (II). II and N-Et-Phe(4-F)-N-Me-Val-N-Et-Tyr(3-tBu)-NHEt showed IC50 of 0.35 and 0.17 nM, resp., for inhibiting binding of 125I-motilin to motilin receptor preparation from mucous membrane of rabbit duodenum.

IT 287205-81-6P

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)

(preparation of peptides or analogs containing substituted phenethylamine moiety as motilin receptor antagonists and drugs for preventing digestive tract movement or high level of blood motilin)

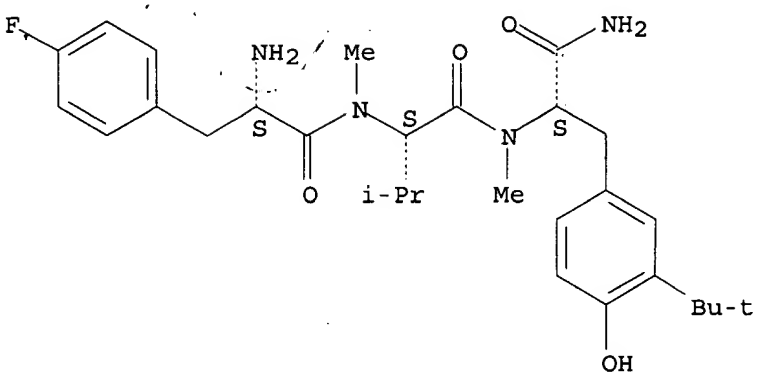
RN 287205-81-6 HCAPLUS

CN L-Tyrosinamide, 4-fluoro-L-phenylalanyl-N-methyl-L-valyl-3-(1,1-dimethylethyl)-Nα-methyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

Updated Search

09890219



REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 3 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2000:210207 HCAPLUS
DOCUMENT NUMBER: 132:251427
TITLE: Preparation of peptide derivatives as motilin receptor antagonists
INVENTOR(S): Matsuoka, Hiroharu; Sato, Tsutomu
PATENT ASSIGNEE(S): Chugai Seiyaku Kabushiki Kaisha, Japan
SOURCE: PCT Int. Appl., 101 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000017231	A1	20000330	WO 1999-JP5215	19990924
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
TW 509699	B	20021111	TW 1999-88116326	19990922
AU 9957592	A1	20000410	AU 1999-57592	19990924
EP 1116726	A1	20010718	EP 1999-944808	19990924
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 3519367	B2	20040412	JP 2000-574139	19990924
US 6586630	B1	20030701	US 2001-787674	20010321
US 2003176643	A1	20030918	US 2003-356558	20030203
US 6720433	B2	20040413		

PRIORITY APPLN. INFO.:
JP 1998-307784 A 19980924
WO 1999-JP5215 W 19990924
US 2001-787674 A3 20010321

OTHER SOURCE(S): MARPAT 132:251427
AB H-Phe-Val-substituted Ala-derivs. represented by general formula

Updated Search

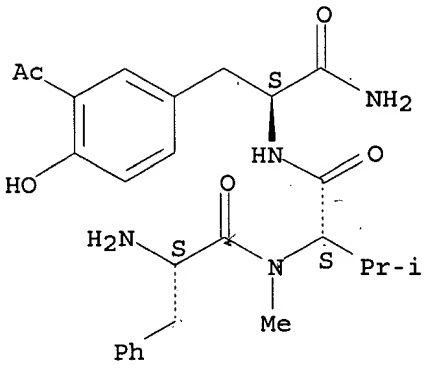
R3CH(CHR1R2)-X-NR4CH(R5)-Y-NR6CH(CH2R8)R7 [R1 = (un)substituted Ph, heterocyclyl, C2-6 linear or branched alkenyl or alkynyl; R2 = H, (un)substituted C1-3 linear or branched alkyl alkyl, NH2, OH; R3 = H, (un)substituted C1-3 linear or branched alkyl, (un)substituted NH2, OH; R4 = H, Me, Et; R5 = (un)substituted C1-6 linear or branched alkyl, C3-7 cycloalkyl, (un)substituted Ph; R6 = H, Me, Et; R7 = H, (un)substituted C1-3 linear or branched alkyl, (un)substituted CONH2; R8 = (un)substituted C3-9 heterocyclyl, (un)substituted Ph], hydrates, or pharmaceutically acceptable salts thereof are prepared Drugs containing these compds. as the active ingredient for motilin receptor antagonists, inhibiting movement of digestive tracts, or treating high level of motilin in blood are also claimed. These peptides are useful for the treatment of irritable bowel syndrome. Thus, Me-Val-Phe(3-tert-butyl-4-F)-NH2 (preparation given) was condensed with Boc-Phe-OH using BOP and diisopropylethylamine in CH2Cl2 at room temperature for 22 h, followed by the treatment with CF3CO2H, to give H-Phe-N-Me-Val-Phe(3-tert-butyl-4-F)-NH2 (I). I showed IC50 of 3.5 nM for inhibiting the binding of [125I]motilin to viscous membrane preparation from rabbit ileum.

IT 262360-77-0P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of peptide derivs. as motilin receptor antagonists and inhibitors of digestive tract motility)

RN 262360-77-0 HCAPLUS

CN L-Tyrosinamide, L-phenylalanyl-N-methyl-L-valyl-3-acetyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 4 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1994:500621 HCAPLUS

DOCUMENT NUMBER: 121:100621

TITLE: Role of endogenous atrial natriuretic peptide in DOCA-salt hypertensive rats: Effects of a novel nonpeptide antagonist for atrial natriuretic peptide receptor

AUTHOR(S): Hirata, Yasunobu; Matsuoka, Hiroaki; Suzuki, Etsu; Hayakawa, Hiroshi; Sugimoto, Tokuichiro; Matsuda, Yuzuru; Morishita, Yoshikazu; Kangawa, Kenji; Minamino, Naoto; et al.

Updated Search

09890219

CORPORATE SOURCE: Second Department Internal Medicine, University Tokyo, Bunkyo, 113, Japan
SOURCE: Circulation (1993), 87(2), 554-61
CODEN: CIRCAZ; ISSN: 0009-7322
DOCUMENT TYPE: Journal
LANGUAGE: English
AB To explore roles of endogenous atrial natriuretic peptide (ANP) in blood pressure and volume regulation, the authors examined the effects of a newly developed ANP antagonist, HS-142-1 (HS) in deoxycorticosterone acetate (DOCA)-salt hypertensive rats. The authors examined the effects of HS on ANP- or brain natriuretic peptide (BNP)-induced redns. in renal vascular resistance (RVR) of rat isolated perfused kidneys; the effects of HS on cGMP production in rat cultured vascular smooth muscle cells pretreated with ANP or BNP; and the renal and systemic effects of HS in DOCA-salt-treated rats and control rats. The authors found that HS dose-dependently reversed ANP- or BNP-induced decreases in RVR and that ANP or BNP at 100 nM caused an 8-fold increase in cGMP production These increases in cGMP were inhibited by HS in a dose-dependent fashion, and 300 μg/mL HS decreased cGMP to the control level. HS alone did not influence RVR or cGMP production DOCA-salt rats showed higher plasma concns. of ANP (198 vs. 75 pg/mL) and BNP (23.7 vs. 2.7 pg/mL) than did the control rats. Bolus administration of 8 mg/kg HS elevated blood pressure by 8%. This rise in blood pressure was attributed to an increase in systemic vascular resistance. Conversely, urinary excretion of sodium (-41%), glomerular filtration rate (-27%), and plasma (-77%) and urinary cGMP (-69%) were decreased by administration of 8 mg/kg HS. These effects were dose-dependent in DOCA-salt rats but slight or negligible in the control rats. These results suggest that endogenous ANP and BNP may be involved in the regulation of blood pressure and body fluid volume in DOCA-salt rats in which ANP and BNP secretion is augmented.
IT 88898-17-3, α-Rat atriopeptin
RL: BIOL (Biological study)
(kidney vascular resistance decrease by)
RN 88898-17-3 HCAPLUS
CN Atrial natriuretic peptide-28 (rat) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L7 ANSWER 5 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1993:248513 HCAPLUS
DOCUMENT NUMBER: 118:248513
TITLE: In vivo and in vitro effects of atrial natriuretic peptide on renin release
AUTHOR(S): Ishimitsu, Toshihiko; Hirata, Yasunobu; Matsuoka, Hiroaki; Ishii, Masao; Sugimoto, Tsuneaki; Kangawa, Kenji; Matsuo, Hisayuki
CORPORATE SOURCE: 2nd Dep. Intern. Med., Univ. Tokyo, Tokyo, 113, Japan
SOURCE: Clinical and Experimental Pharmacology and Physiology (1992), 19(10), 711-16
CODEN: CEXPB9; ISSN: 0305-1870
DOCUMENT TYPE: Journal
LANGUAGE: English
AB This study investigated the effect of atrial natriuretic peptide on renin release from the kidney. The in vitro effect was examined using rat renal cortical slices, and the in vivo effect was observed in a human infusion study. In the in vitro expts., α-human atrial natriuretic peptide (α-hANP) (10-9-10-6 mol/L) did not change the basal renin release rate from the renal cortical slices. Isoproterenol (10-6 mol/L) increased renin release by 40%, whereas angiotensin II (10-6 mol/L) suppressed it by

48%. However, α-hANP failed to change the plasma renin activity in normotensive subjects or patients with essential hypertension, or even in patients with raised renin levels such as renovascular hypertension or congestive heart failure. Apparently, atrial natriuretic peptide is not involved in renin release from the juxtaglomerular apparatus

IT 89213-87-6
RL: BIOL (Biological study)
(renin release response to, in human)
RN 89213-87-6 HCAPLUS
CN Atrial natriuretic peptide-28 (human) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L7 ANSWER 6 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1991:551687 HCAPLUS
DOCUMENT NUMBER: 115:151687
TITLE: Nephrogenous cyclic GMP production during sodium chloride loading and ANP infusion
AUTHOR(S): Hirata, Yasunobu; Fukui, Kazushige; Hayakawa, Hiroshi; Namba, Shinichiro; Ishimitsu, Toshihiko; Sugimoto, Tokuichiro; Kimura, Kenjiro; Matsuoka, Hiroaki ; Sugimoto, Tsuneaki
CORPORATE SOURCE: Fac. Med., Univ. Tokyo, Tokyo, 113, Japan
SOURCE: Japanese Heart Journal (1990), 31(6), 809-16
CODEN: JHEJAR; ISSN: 0021-4868
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Six normotensive (NTs) and 7 essential hypertensives (HTs) were placed on 7-day low (3 g/day) and then 7-day high NaCl diets (20 g/day). On the last day of each period, the natriuretic and nephrogenous cGMP responses to atrial natriuretic peptide (ANP) infusion at 25 ng/kg/min for 40 min were determined ANP infusion markedly increased the plasma concns. of ANP and cGMP and the urinary excretions of Na and cGMP. These changes were accompanied by a rise in nephrogenous cGMP. Increases in nephrogenous cGMP during ANP infusion were not different between HTs and NTs despite a greater natriuretic response in HTs. NaCl loading increased the natriuretic response to ANP infusion in both groups. However, nephrogenous cGMP production induced by ANP infusion was not affected by changes in NaCl intake. Thus, although ANP-induced natriuresis is associated with an increase in nephrogenous cGMP, the natriuretic effect of ANP seems to be modified to a greater extent by indirect mechanisms such as renal perfusion pressure and body fluid volume status.

IT 89213-87-6
RL: BIOL (Biological study)
(cGMP formation response to, in kidney in human, sodium chloride loading effect on)
RN 89213-87-6 HCAPLUS
CN Atrial natriuretic peptide-28 (human) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L7 ANSWER 7 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1991:75781 HCAPLUS
DOCUMENT NUMBER: 114:75781
TITLE: Effects of atrial natriuretic peptide on renal arterioles: morphometric analysis using microvascular casts
AUTHOR(S): Kimura, Kenjiro; Hirata, Yasunobu; Nanba, Shinichiro; Tojo, Akihiro; Matsuoka, Hiroaki; Sugimoto,

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Tsuneaki
CORPORATE SOURCE: Fac. Med., Univ. Tokyo, Tokyo, 113, Japan
SOURCE: American Journal of Physiology (1990), 259(6, Pt. 2), F936-F944
CODEN: AJPHAP; ISSN: 0002-9513
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In normal rat kidneys, the effect of atrial natriuretic peptide (ANP) on the diameter of the arterioles was evaluated by SEM of vascular casts. Acryl resin was infused into rat kidneys during the administration of ANP, either alone or with norepinephrine (NE). ANP infusion constricted the proximal efferent arteriole in the superficial cortex. Although NE constricted the proximal and distal segments of the afferent arteriole in the superficial cortex, the addition of ANP reversed the constriction and further constricted the efferent arteriole. In the deep cortex, only the proximal segment of the afferent arteriole was dilated by ANP when infused with NE. In a sep. set of expts., ANP increased both the glomerular filtration rate (GFR) and urinary Na excretion (UNaV), and NE decreased the renal blood flow (RBF). However, administration of ANP after NE recovered RBF and increased GFR as well as UNaV. The results indicate that ANP increases GFR and natriuresis by constricting the efferent arteriole. NE appears to decrease RBF by constricting the afferent arteriole. ANP antagonizes the renal effects of NE primarily by dilating afferent arterioles.

IT 88898-17-3
RL: BIOL (Biological study)
(kidney arterioles response to, norepinephrine in relation to)

RN 88898-17-3 HCAPLUS
CN Atrial natriuretic peptide-28 (rat) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L7 ANSWER 8 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1990:471837 HCAPLUS
DOCUMENT NUMBER: 113:71837
TITLE: Effects of chronic administration of atrial natriuretic polypeptide on glomerular lesions in spontaneously hypertensive rats
AUTHOR(S): Kimura, Kenjiro; Drozdova, G.; Hirata, Yasunobu; Matsuoka, Hiroaki; Ishii, Masao; Sugimoto, Tsuneaki; Kangawa, Kenji; Matsuo, Hisayuki
CORPORATE SOURCE: Fac. Med., Univ. Tokyo, Tokyo, 113, Japan
SOURCE: Japanese Heart Journal (1990), 31(2), 227-36
CODEN: JHEJAR; ISSN: 0021-4868
DOCUMENT TYPE: Journal
LANGUAGE: English

AB To investigate the effects of atrial natriuretic polypeptides (ANP) on hypertensive glomerular lesions, ANP was administered i.v. by osmotic minipumps to 15-wk-old male spontaneously hypertensive rats (SHRs) in a sustained hypertensive stage. ANP was infused at the rate of 100 ng/h/rat (the ANP group). Saline was similarly administered to age-matched SHRs (the control group). The rats were sacrificed on the 7th day. Semiquant. evaluation of the renal tissue revealed no significant difference in glomerular sclerosis between the 2 groups. However, segmental hyalinosis in glomeruli was more accentuated in the ANP group than in the control group. Hyalinosis in glomeruli may be related to the elevated intracapillary pressure, so these results are in accordance with reports that ANP increases glomerular capillary pressure by preglomerular vasodilation and postglomerular vasoconstriction. It remains to be determined

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whether endogenous ANP works as an aggravating factor for glomerular injuries in the natural course of hypertension.

IT 88898-17-3
RL: BIOL (Biological study)
(kidney glomerulus lesions in hypertension response to)
RN 88898-17-3 HCAPLUS
CN Atrial natriuretic peptide-28 (rat) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L7 ANSWER 9 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1989:626021 HCAPLUS
DOCUMENT NUMBER: 111:226021
TITLE: Renal vasoconstriction by the endothelial cell-derived peptide endothelin in spontaneously hypertensive rats
AUTHOR(S): Hirata, Yasunobu; Matsuoka, Hiroaki; Kimura, Kenjiro; Fukui, Kazushige; Hayakawa, Hiroshi; Suzuki, Etsu; Sugimoto, Tokuichiro; Sugimoto, Tsuneaki; Yanagisawa, Masashi; Masaki, Tomoh
CORPORATE SOURCE: Fac. Med., Univ. Tokyo, Tokyo, 113, Japan
SOURCE: Circulation Research (1989), 65(5), 1370-9
CODEN: CIRUAL; ISSN: 0009-7330
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effects of endothelin were examined on systemic and renal hemodynamics in anesthetized spontaneously hypertensive rats (SHR) and Wistar-Kyoto rats. Endothelin (500 ng i.v. + 1000 ng/h/300-g rat) elevated mean blood pressure by 13% and decreased renal blood flow by 71% and glomerular filtration rate by 66%, resulting in a 430% increase in renal vascular resistance (RVR) in SHR. This rise in blood pressure was associated with an increase in hematocrit (+8%), but a decrease in urinary Na excretion (-51%). This dose of endothelin reduced cardiac output by 40% and brought about a 96% rise in systemic vascular resistance (SVR). However, the SVR increase was smaller than the RVR increase. These changes in systemic and renal hemodynamics were observed in a dose-dependent manner, and the degrees of change did not differ between the 2 strains. Addnl. infusion of atrial natriuretic peptide (0.33 μg/kg/min) into SHR completely reversed the changes in blood pressure and renal hemodynamics caused by endothelin, resulting in pronounced natriuresis (+760%). Renal vascular casting revealed that endothelin mainly constricted the arcuate and interlobular arteries, as well as afferent arterioles. Thus, endothelin may be involved in blood pressure and body fluid volume regulation through systemic and renal vasoconstriction.

IT 88898-17-3
RL: BIOL (Biological study)
(endothelin effect on blood pressure and renal hemodynamics in hypertension reversal by)
RN 88898-17-3 HCAPLUS
CN Atrial natriuretic peptide-28 (rat) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L7 ANSWER 10 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1987:418501 HCAPLUS
DOCUMENT NUMBER: 107:18501
TITLE: Evidence for lack of a role of cGMP in effect of α-hANP on aldosterone inhibition
AUTHOR(S): Matsuoka, Hiroaki; Ishii, Masao; Hirata, Yasunobu; Atarashi, Keiichiro; Sugimoto, Tsuneaki;

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Kangawa, Kenji; Matsuo, Hisayuki
CORPORATE SOURCE: 2nd Dep. Intern. Med., Univ. Tokyo, Tokyo, Japan
SOURCE: American Journal of Physiology (1987), 252(5, Pt. 1), E643-E647
CODEN: AJPHAP; ISSN: 0002-9513
DOCUMENT TYPE: Journal
LANGUAGE: English
AB To investigate the role of cGMP in the inhibitory effect on aldosterone production of α-human atrial natriuretic polypeptide (α-hANP), the effects of the peptide were compared with those of sodium nitroprusside (SNP) on the production of aldosterone and cGMP in dispersed adrenal capsular cells of rats, the effects of derivs. of cGMP were examined on the production of aldosterone, and the influence of K on the effects of α-hANP on the production of aldosterone and cGMP was studied. α-HANP (3 + 10-8-3 + 10-7 M) decreased the production of aldosterone in a dose-dependent manner, and markedly increased the production of cGMP. On the other hand, although SNP at 10-5-10-3 M increased the production of cGMP in a dose-dependent manner, it caused no changes in the production of aldosterone. Neither dibutyryl cGMP nor 8-bromo-cGMP affected the production of aldosterone in the adrenal cells. Although the aldosterone-inhibitory effect of α-hANP was lost in the K-free medium, the cGMP-stimulatory effect of the peptide was not altered by adding K to the incubation medium at concns. of 0-5 mequiv/L. Apparently, cGMP plays a minor role in the inhibitory effect of α-hANP on the production of aldosterone, and the production of cGMP stimulated by the peptide is not directly involved in the decrease in aldosterone production in adrenal capsular cells of rats.
IT 89213-87-6
RL: BIOL (Biological study)
(aldosterone formation inhibition by, cGMP in relation to)
RN 89213-87-6 HCAPLUS
CN Atrial natriuretic peptide-28 (human) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L7 ANSWER 11 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1987:189821 HCAPLUS
DOCUMENT NUMBER: 106:189821
TITLE: Blood pressure, renal and endocrine responses to α-human atrial natriuretic polypeptide in healthy volunteers
AUTHOR(S): Ishii, Masao; Sugimoto, Tokuichiro; Matsuoka, Hiroaki; Ishimitsu, Toshihiko; Atarashi, Keiichiro; Hirata, Yasunobu; Sugimoto, Tsuneaki; Kangawa, Kenji; Matsuo, Hisayuki
CORPORATE SOURCE: 2nd Dep. Intern. Med., Univ. Tokyo, Tokyo, 113, Japan
SOURCE: Japanese Heart Journal (1986), 27(6), 777-89
CODEN: JHEJAR; ISSN: 0021-4868
DOCUMENT TYPE: Journal
LANGUAGE: English
AB I.v. infusion of graded doses of α-human atrial natriuretic polypeptide (α-hANP) [89213-87-6] resulted in a dose-dependent decrease in blood pressure and an increase in heart rate in healthy male volunteers. However, there were no changes in urine output or in the urinary excretion rate of Na. Glomerular filtration rate did not change, whereas renal blood flow decreased, leading to increases in filtration fraction and renal vascular resistance. Although plasma renin [9015-94-5] activity and plasma concentration of norepinephrine [51-41-2] increased during infusion of α-hANP, plasma concns. of aldosterone

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[52-39-1] and cortisol [50-23-7] decreased. Plasma concentration of AVP [113-79-1] did not change during the infusion, but greatly increased after cessation of the infusion. The hematocrit increased during the infusion. Although α-hANP has a potent hypotensive action and inhibits the secretion of aldosterone, cortisol, and probably AVP, it does not dilate renal vessels in normotensive persons, and likely increases vascular permeability. The lack of consistent diuretic and natriuretic responses to α-hANP may be related to the predominance of the hypotensive effect over the renal effects of the peptide in normotensive persons, or a diurnal change may have served to obscure such a response.

IT 89213-87-6, α-Human atrial natriuretic polypeptide
RL: BIOL (Biological study)
(cardiovascular and endocrine and kidney responses to, in men)
RN 89213-87-6 HCAPLUS
CN Atrial natriuretic peptide-28 (human) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L7 ANSWER 12 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1986:162495 HCAPLUS
DOCUMENT NUMBER: 104:162495
ORIGINAL REFERENCE NO.: 104:25569a,25572a
TITLE: Effects of α-human atrial natriuretic polypeptide (α-hANP) on the main regulatory mechanisms for blood pressure and body fluid volume in rats
AUTHOR(S): Ishii, Masao; Matsuoka, Hiroaki; Hirata, Yasunobu; Sugimoto, Tokuichiro; Ishimitsu, Toshihiko; Sugimoto, Tsuneaki; Kanagawa, Kenji; Matsuo, Hisayuki
CORPORATE SOURCE: Second Dep. Intern. Med., Univ. Tokyo, Tokyo, 113, Japan
SOURCE: Japanese Circulation Journal (1985), 49(9), 969-72
CODEN: JCIRA2; ISSN: 0047-1828
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In anesthetized male rats, synthetic α-human atrial natiuretic polypeptide (α-hANP) [88898-17-3] had a potent hypotensive action when administered i.v. (1-8 μg/300 g). α-HANP also exhibited marked diuretic and natriuretic properties.. The hypotensive action was attributed to both the dilation of resistant vessels and a decrease in cardiac output. α-HANP (3 + 10-2-10-7 M) dose-dependently inhibited aldosterone [52-39-1] production by dispersed adrenal capsular cells of female rats. CAMP [60-92-4] production was inhibited and cGMP [7665-99-8] production was stimulated by 10-7-10-6 M α-hANP in this system. α-HANP (10-9 and 10-2 M) also inhibited renin [9015-94-5] secretion by rat renal slices. Thus, α-hANP acts on systems regulating blood pressure and body fluid volume by modulating interactions between the heart and kidney.

IT 88898-17-3
RL: BIOL (Biological study)
(blood pressure and body fluid regulation by, mechanisms for)
RN 88898-17-3 HCAPLUS
CN Atrial natriuretic peptide-28 (rat) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L7 ANSWER 13 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1985:572962 HCAPLUS
DOCUMENT NUMBER: 103:172962

09890219

ORIGINAL REFERENCE NO.: 103:27679a,27682a
TITLE: The effects of human atrial 28-amino acid peptide on systemic and renal hemodynamics in anesthetized rats
AUTHOR(S): Hirata, Yasunobu; Ishii, Masao; Sugimoto, Tokuichiro; Matsuoka, Hiroaki; Sugimoto, Tsuneaki; Kangawa, Kenji; Matsuo, Hisayuki
CORPORATE SOURCE: 2nd. Dep. Intern. Med., Univ. Tokyo Hosp., Tokyo, 113, Japan
SOURCE: Circulation Research (1985), 57(4), 634-9
CODEN: CIRUAL; ISSN: 0009-7330
DOCUMENT TYPE: Journal
LANGUAGE: English
AB α-Human atrial natriuretic polypeptide (I) [89213-87-6] infusion (0.67 μg/kg/min) reduced mean arterial pressure, cardiac output, and total peripheral resistance by 21, 9, and 11%, resp., after 10 min of infusion in anesthetized rats. Despite the marked decrease in blood pressure, the heart rate did not change. Increases in urine volume, urinary Na excretion, filtration fraction, and fractional Na excretion and a decrease in renal vascular resistance were dose dependent: +490%, +1340%, +19%, +1160%, and -18%, resp., with 0.67 μg/kg/min of I. The glomerular filtration rate increased with 0.33 and 0.67 μg/kg/min of I, whereas renal blood flow did not change. Changes in urinary Na excretion and fractional Na excretion were highly correlated, but changes in urinary Na excretion were not correlated with changes in the glomerular filtration rate. The dose-related changes in renal vascular resistance during infusion of I solns. did not differ from those in total peripheral resistance. Evidently, the hypotensive effect of I is attributed to decreases in total peripheral resistance and cardiac output. That I dilates renal resistance vessels as well as systemic resistance vessels, and that diuresis and natriuresis are induced by I are attributed to increased glomerular filtration rate and to inhibition of tubular Na reabsorption.
IT 89213-87-6
RL: BIOL (Biological study)
(heart and kidney of laboratory animal hemodynamics response to)
RN 89213-87-6 HCAPLUS
CN Atrial natriuretic peptide-28 (human) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L7 ANSWER 14 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1985:432467 HCAPLUS
DOCUMENT NUMBER: 103:32467
ORIGINAL REFERENCE NO.: 103:5171a,5174a
TITLE: Effect of α-human atrial natriuretic polypeptide on aldosterone secretion from the dispersed adrenal cells of rat
AUTHOR(S): Matsuoka, Hiroaki; Ishii, Tomio; Sugimoto, Tsuneaki; Kangawa, Kenji; Matsuo, Hisayuki
CORPORATE SOURCE: Sch. Med., Univ. Tokyo, Tokyo, Japan
SOURCE: Horumon to Rinsho (1985), 33(4), 359-63
CODEN: HORIAE; ISSN: 0439-5875
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB Production of aldosterone (I) [52-39-1] by a suspension of adrenal capsular cells from rats (SD strain) was decreased by 30 nM human α-atrial natriuretic polypeptide (α-hANP) [89213-87-6] from 22.6 to 16.9 and by 1 mM ouabain (II) [630-60-4] from 21.5 to 5.9 ng/106 cells/h. Higher concns. of II gave rise to an increase in I production from

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21.5 to 33.0 ng/106 cells/h. The α-hANP (>0.1 mM) slightly inhibited cAMP [60-92-4] formation by the adrenal capsular cells, whereas α-hANP (>1 nM) accelerated cGMP [7665-99-8] production Participation of I in α-hANP-induced diuresis and a physiol. regulatory effect of α-hANP in I secretion are suggested.

IT 89213-87-6
RL: BIOL (Biological study)
(aldosterone secretion response to, mechanism for)

RN 89213-87-6 HCAPLUS
CN Atrial natriuretic peptide-28 (human) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L7 ANSWER 15 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1985:179796 HCAPLUS
DOCUMENT NUMBER: 102:179796
ORIGINAL REFERENCE NO.: 102:28115a,28118a
TITLE: Inhibition of aldosterone production by α-human atrial natriuretic polypeptide is associated with an increase in cGMP production
AUTHOR(S): Matsuoka, Hiroaki; Ishii, Masao; Sugimoto, Tokuichiro; Hirata, Yasunobu; Sugimoto, Tsuneaki; Kangawa, Kenji; Matsuo, Hisayuki
CORPORATE SOURCE: Fac. Med., Univ. Tokyo, Tokyo, 113, Japan
SOURCE: Biochemical and Biophysical Research Communications (1985), 127(3), 1052-6
CODEN: BBRCA9; ISSN: 0006-291X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Synthetic α-human atrial natriuretic polypeptide [89213-87-6] caused rapid and marked inhibition of aldosterone [52-39-1] production in dispersed rat adrenal capsular cells. The polypeptide also slightly, but significantly, decreased cAMP [60-92-4] production in the adrenal dispersed capsular cells, while markedly stimulating cGMP [7665-99-8] production The cGMP production was accelerated at a concentration of α-human atrial natriuretic polypeptide lower than the threshold level to stimulate aldosterone production Apparently, α-human atrial natriuretic polypeptide plays a regulatory role in aldosterone production and an addnl. role in natriuresis through inhibition of aldosterone production The stimulation of cGMP production by α-human atrial natriuretic polypeptide may be involved in the inhibitory effect of this peptide on aldosterone production

IT 89213-87-6
RL: BIOL (Biological study)
(aldosterone and cGMP formation by adrenal cortex response to)

RN 89213-87-6 HCAPLUS
CN Atrial natriuretic peptide-28 (human) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

=> d his

(FILE 'HOME' ENTERED AT 19:45:51 ON 30 JAN 2008)

FILE 'REGISTRY' ENTERED AT 19:47:05 ON 30 JAN 2008
L1 STRUCTURE UPLOADED
L2 50 S L1

Updated Search

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L3 STRUCTURE UPLOADED
L4 50 S L3
L5 58230 S L3 FULL

FILE 'HCAPLUS' ENTERED AT 19:55:28 ON 30 JAN 2008
L6 21413 S L5
L7 15 S L6 AND MATSUOKA, H?/AU

=> s l6 not l7
L8 21398 L6 NOT L7

=> s l8 and sato, t?/au
24961 SATO, T?/AU
L9 26 L8 AND SATO, T?/AU

=> d l9, ibib abs fhitstr, 1-26

L9 ANSWER 1 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2006:607454 HCAPLUS
DOCUMENT NUMBER: 145:76923
TITLE: Involvement of spinal μ1-opioid receptors on the Tyr-D-Arg-Phe-sarcosine-induced antinociception
AUTHOR(S): Mizoguchi, Hirokazu; Nakayama, Daisuke; Watanabe, Hiroyuki; Ito, Kanenori; Sakurada, Wataru; Sawai, Toshiki; Fujimura, Tsutomu; Sato, Takumi; Sakurada, Tsukasa; Sakurada, Shinobu
CORPORATE SOURCE: Department of Physiology and Anatomy, Tohoku Pharmaceutical University, 4-4-1 Komatsushima, Aoba-ku, Sendai, 981-8558, Japan
SOURCE: European Journal of Pharmacology (2006), 540(1-3), 67-72
CODEN: EJPHAZ; ISSN: 0014-2999
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The involvement of spinal μ-opioid receptor subtypes on the antinociception induced by i.t.-administered Tyr-D-Arg-Phe-sarcosine (TAPS), a N-terminal tetrapeptide analog of dermorphin, was determined in mice tail-flick test. Intrathecal administration of TAPS produced the marked inhibition of the tail-flick response in a dose-dependent manner. The antinociception induced by TAPS was completely eliminated by i.t.-co-administration of Tyr-D-Pro-Phe-Phe-NH2 (D-Pro2-endomorphin-2), the μ1-opioid receptor antagonist, whereas i.t. co-treatment with Tyr-D-Pro-Trp-Phe-NH2 (D-Pro2-endomorphin-1) or Tyr-D-Pro-Trp-Gly-NH2 (D-Pro2-Tyr-W-MIF-1), the μ2-opioid receptor antagonists, did not affect the TAPS-induced antinociception. In contrast, the antinociception induced by i.t.-administered [D-Ala2,N-MePhe4,Gly-ol5]enkephalin was significantly attenuated by i.t.-co-administration of D-Pro2-endomorphin-1 or D-Pro2-Tyr-W-MIF-1, but not D-Pro2-endomorphin-2. These results suggest that TAPS may stimulate spinal μ1-opioid receptors to produce the antinociception.
IT 90549-86-3, Tyr-D-Arg-Phe-sarcosine
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Tyr-D-Arg-Phe-sarcosine-induced antinociception via spinal μ1-opioid receptors)
RN 90549-86-3 HCAPLUS
CN Glycine, L-tyrosyl-D-arginyl-L-phenylalanyl-N-methyl- (CA INDEX NAME)

Updated Search

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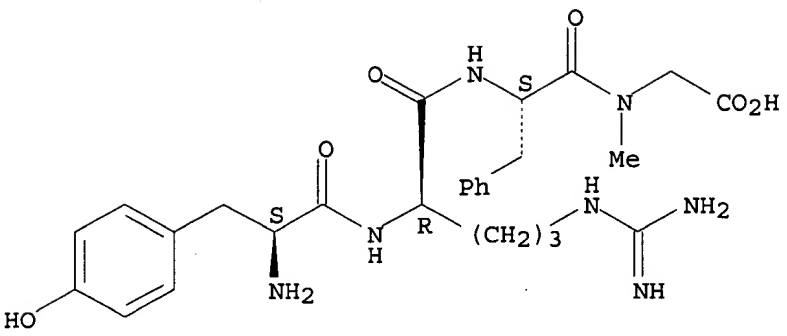
Absolute stereochemistry.



REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 2 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2005:1012141 HCAPLUS
DOCUMENT NUMBER: 143:435206
TITLE: A Peptide Motif Recognizing a Polymer Stereoregularity
AUTHOR(S): Serizawa, Takeshi; Sawada, Toshiki; Matsuno, Hisao; Matsubara, Teruhiko; Sato, Toshinori
CORPORATE SOURCE: Research Center for Advanced Science and Technology (RCAST), University of Tokyo, 4-6-1 Komaba, Meguro-ku, Tokyo, 153-8904, Japan
SOURCE: Journal of the American Chemical Society (2005), 127(40), 13780-13781
CODEN: JACSAT; ISSN: 0002-7863
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A combinatorial phage display method was applied to films composed of a stereoregular polymer of methacrylates. The phage clones with selective affinity for isotactic (it) poly(Me methacrylate) (PMMA) were isolated. Greater amts. of the phage clones bound to it-PMMA, compared to other stereoregular PMMAs. The phage expressing ELWRPTR most strongly bound to the polymer, and the selectivity was also the best. The peptide motif essential for the specific interaction with the stereoregular polymer was revealed.

IT 850015-56-4P
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); BUU (Biological use, unclassified); CPN (Combinatorial preparation); ANST (Analytical study); BIOL (Biological study); CMBI (Combinatorial study); PREP (Preparation); USES (Uses)
(peptide motif recognizing polymer stereoregularity of isotactic poly(Me methacrylate))

RN 850015-56-4 HCAPLUS
CN L-Phenylalanine, L-asparaginyl-L-leucyl-L-glutaminyl-L-α-glutamyl-L-phenylalanyl-L-leucyl- (CA INDEX NAME)

Absolute stereochemistry.

Updated Search

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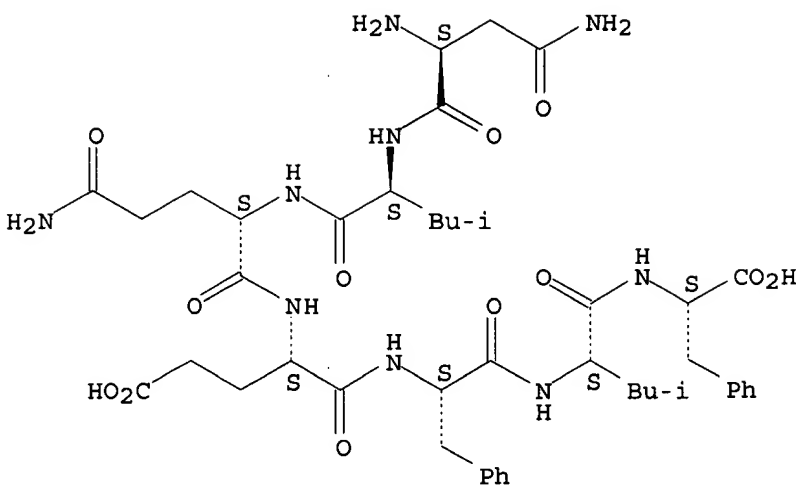


REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

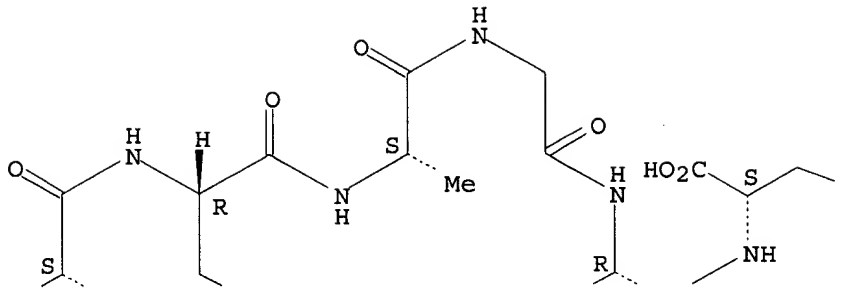
L9 ANSWER 3 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2005:576093 HCAPLUS
DOCUMENT NUMBER: 144:287703
TITLE: Tertiary structure-function relationship of highly disulfide-bridged peptides
AUTHOR(S): Sato, Takashi
CORPORATE SOURCE: Department of Applied Biological Sciences, Saga University, Saga, 840-8502, Japan
SOURCE: Peptide Science (2005), Volume Date 2004, 41st, 677-680
CODEN: PSCIFQ; ISSN: 1344-7661
PUBLISHER: Japanese Peptide Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Tertiary structure function relationship of heat-stable enterotoxin (ST) was investigated. The tertiary structures of fully toxic, partially toxic and nontoxic analogs were revealed by both methods of NMR study and x-ray crystallog. Mol. recognition of ST was interpreted based on those fine structures in terms of receptor binding affinities and toxicities. Mechanisms for expression of toxic activity were suggested by assembled structure of this mol.
IT 878481-75-5
RL: ADV (Adverse effect, including toxicity); PRP (Properties); BIOL (Biological study)
(tertiary structure-function relationship of highly disulfide-bridged peptides)
RN 878481-75-5 HCAPLUS
CN L-Tyrosine, L-asparaginyl-L-phenylalanyl-L-threonyl-L-tyrosyl-L-cysteinyl-L-cysteinyl-L-α-glutamyl-L-leucyl-L-cysteinyl-L-cysteinyl-L-asparaginyl-L-prolyl-L-alanyl-L-cysteinyl-L-alanylglycyl-L-cysteinyl-, cyclic (5→10),(6→14),(9→17)-tris(disulfide) (9CI) (CA INDEX NAME)

Absolute stereochemistry.

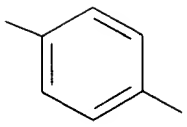
Updated Search

09890219

PAGE 1-A

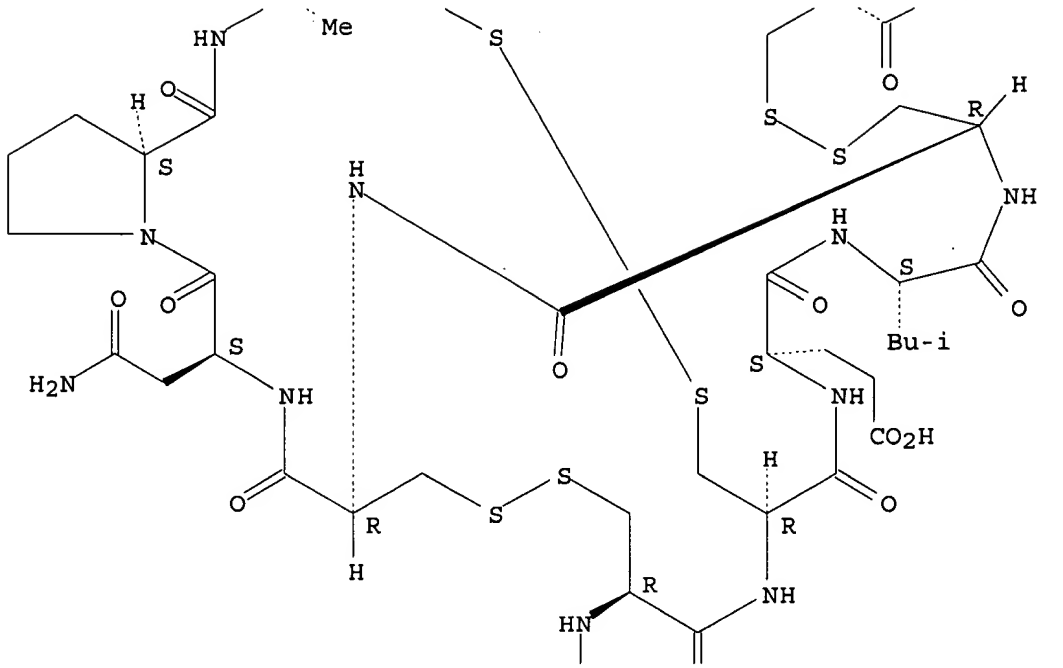


PAGE 1-B



Updated Search

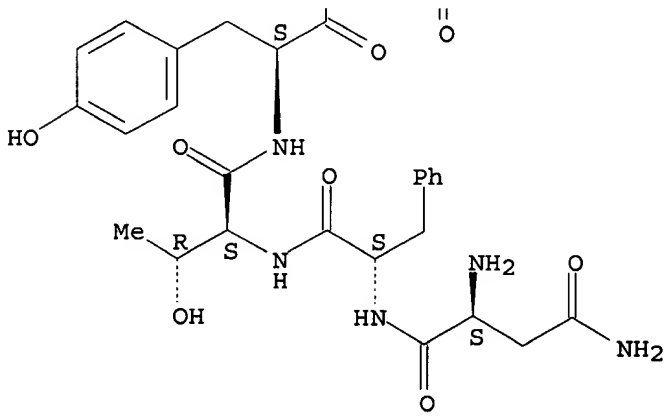
PAGE 2-A



PAGE 2-B

OH

PAGE 3-A



REFERENCE COUNT: 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 4 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:899999 HCAPLUS
DOCUMENT NUMBER: 142:51620

Updated Search

09890219

TITLE: Efficient in-gel digestion procedure using 5-cyclohexyl-1-pentyl-β-D-maltoside as an additive for gel-based membrane proteomics

AUTHOR(S): Katayama, Hiroyuki; Tabata, Tsuyoshi; Ishihama, Yasushi; Sato, Toshitaka; Oda, Yoshiya; Nagasu, Takeshi

CORPORATE SOURCE: Laboratory of Seeds Finding Technology, Eisai Co. Ltd., Tsukuba, 300-2635, Japan

SOURCE: Rapid Communications in Mass Spectrometry (2004), 18(20), 2388-2394
CODEN: RCMSEF; ISSN: 0951-4198

PUBLISHER: John Wiley & Sons Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB A cycloalkyl aliphatic saccharide, 5-cyclohexyl-1-pentyl-β-D-maltoside (CYMAL-5), was evaluated as a novel additive in a high-throughput in-gel protein digestion system using 96-well plates. Addition of 0.1% CYMAL-5 (final concentration) during trypsin treatment was compatible with both matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOFMS) and liquid chromatog./tandem mass spectrometry (LC/MS/MS) anal., and gave a better digestion efficiency than n-octylglucoside, which we previously reported. In-gel reduction and alkylation of Cys residues under denaturing conditions also improved the sequence coverage of peptides. In-gel tryptic digestion with the optimum combination of 0.5 mm thick gels, neg. staining, alkylation under denaturing conditions (6 M guanidine hydrochloride), and digestion in the presence of CYMAL-5, gave excellent performance especially for membrane protein anal., where recovery of hydrophobic peptides was markedly enhanced. The new protocol is simple and convenient, and should be widely applicable to gel-based proteomics.

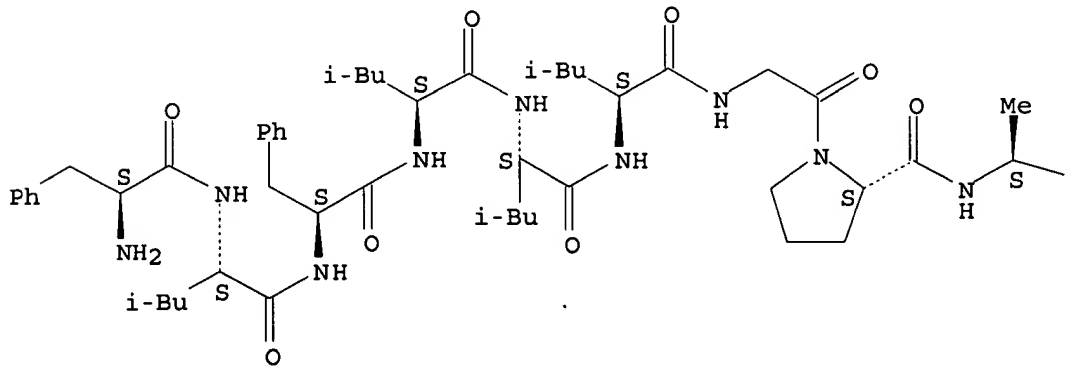
IT 809241-37-0
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(efficient in-gel digestion procedure using 5-cyclohexyl-1-pentyl-β-D-maltoside as additive for gel-based membrane proteomics)

RN 809241-37-0 HCAPLUS

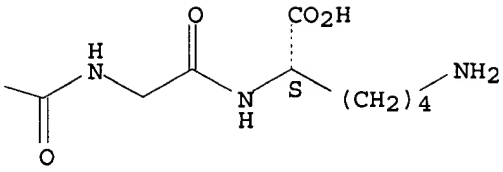
CN L-Lysine, L-phenylalanyl-L-leucyl-L-phenylalanyl-L-leucyl-L-leucyl-L-leucylglycyl-L-prolyl-L-alanylglycyl- (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



Updated Search



REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 5 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2002:160176 HCAPLUS
DOCUMENT NUMBER: 136:227961
TITLE: DNA and protein sequences of four Lentinula edodes laccase sequence homologs and their uses
INVENTOR(S): Sato, Toshitsugu; Hirano, Tatsuya; Watanabe, Hisataka; Ei, Hitoshi
PATENT ASSIGNEE(S): Iwate Prefecture, Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 30 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

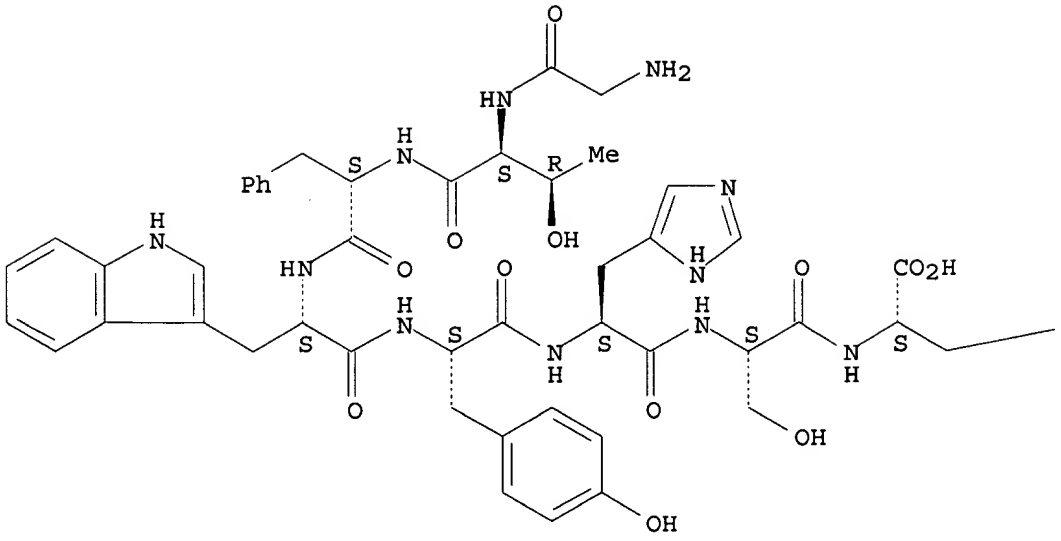
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2002065282	A	20020305	JP 2000-267473	20000904
PRIORITY APPLN. INFO.:			JP 2000-267473	20000904

AB This invention provides four laccase sequence homologs (lcc1, lcc2, lcc2' and lcc3) cloned from Lentinula edodes. Sequence anal. showed that the Lentinula edodes laccase sequence homologs contains 10 copper binding site and the sequence of these enzymes have high homolog with their counterparts from Agaricus bisporus and pleurotus ostreatus. The laccase can be used for waste treatment in papermaking industry.

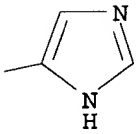
IT 402933-30-6
RL: PRP (Properties)
(unclaimed sequence; dNA and protein sequences of four Lentinula edodes laccase sequence homologs and their uses)
RN 402933-30-6 HCAPLUS
CN L-Histidine, glycyl-L-threonyl-L-phenylalanyl-L-tryptophyl-L-tyrosyl-L-histidyl-L-seryl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 6 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2002:90066 HCAPLUS
DOCUMENT NUMBER: 136:135034
TITLE: Method for producing tripeptide derivative
INVENTOR(S): Sato, Tsutomu; Shimizu, Hirohito
PATENT ASSIGNEE(S): Chugai Seiyaku Kabushiki Kaisha, Japan
SOURCE: PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

Updated Search

09890219

WO 2002008248 A1 20020131 WO 2001-JP6295 20010719
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT,
RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US,
UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
JP 2005097119 A 20050414 JP 2000-219977 20000721
PRIORITY APPLN. INFO.: JP 2000-219977 A 20000721
OTHER SOURCE(S): CASREACT 136:135034; MARPAT 136:135034
GI

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

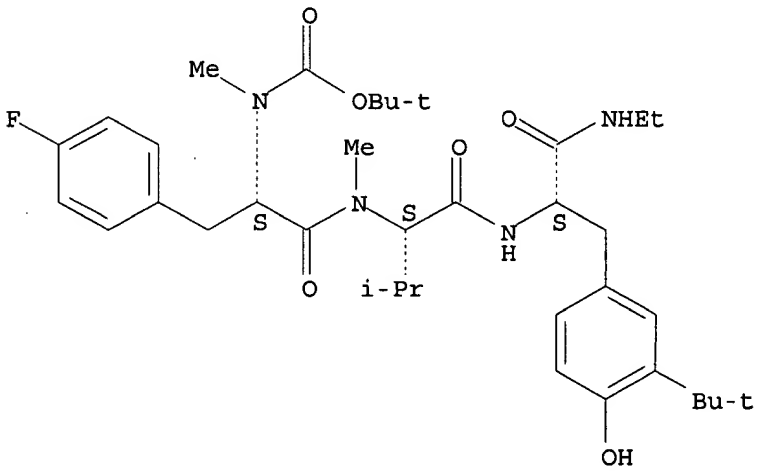
AB A method for producing L-phenylalanyl-L-valyl-L-3-tert-butyl-L-tyrosinamide compds. represented by the general formula (I; wherein R1 represents a hydrogen atom or a linear or branched aliphatic alkyl group having 1 to 4 carbon atoms; R2 represents a hydrogen atom or Me group; R3 represents a hydrogen atom or Me group; and R4 represents a halogen atom) comprises condensation of 3-tert-butyl-L-tyrosinamide derivs. (II; R1 , R2 = same as above) with N-methyl-L-valine derivs. (III; P1 = amino-protecting group), N-deprotection of the resulting L-valyl-3-tert-butyl-L-tyrosinamide derivs. (IV; R1, R2, P1 = same as above), and condensation of the resulting IV (P1 = H; R1 , R2 = same as above) with L-phenylalanine derivs. (V; R3, R4 = same as above; P2 = amino-protecting group) followed by N-deprotection. The method can be advantageously used for producing a novel peptide derivative in a com. process. Thus, 20.8 g MeSO3H and 20.0 g tert-Bu chloride were successively added to 10.0 g L-tyrosine Me ester hydrochloride under stirring, stirred at 50° for 5 h, treated dropwise with MeOH (20 mL)/H2O (20 mL) under ice-cooling then with a solution of 14.2 g KOH in 43 mL H2O at <10° to give 77.0% 3-tert-butyl-L-tyrosine Me ester which (8.35 g) was added to a mixture of 24.1 g 62% aqueous ethylamine and 7.52 g ethylamine hydrochloride under ice-cooling and stirred at room temperature for 5 h to give 89.8% 3-tert-butyl-L-tyrosine ethylamide (VI). To a solution of 5.50 g VI and 3.35 g 1-hydroxybenzotriazole monohydrate in 55 mL THF were successively added 4.19 g 1-ethyl-3-(3-dimethylaminopropyl)carbodiimide hydrochloride and 3.04 mL Et3N and stirred at room temperature for 2.5 h to give 100% N-tert-butoxycarbonyl-N-methyl-L-valyl-3-tert-butyl-L-tyrosine ethylamide which (10.0 g) was dissolved in 100 mL EtOAc, treated with 11.1 mL concentrated H2SO4 under ice-cooling, treated with 100 mL EtOAc, adjusted pH 8 by adding saturated aqueous NaHCO3, and stirred 15 min to give 87.9% N-methyl-L-valyl-3-tert-butyl-L-tyrosine ethylamide (VII). To a mixture of 5.50 g VII, 5.20 g N-tert-butoxycarbonyl-N-methyl-4-fluoro-L-phenylalanine, 4.47 g 2-chloro-1-methylpyridinium iodide, and 37 mL tert-Bu Me ether was added 5.09 mL Et3N and stirred at room temperature for 4 h to give 86.0% N-tert-butoxycarbonyl-N-methyl-4-fluoro-L-phenylalanyl-N-methyl-L-valyl-3-tert-butyl-L-tyrosine ethylamide which (7.50 g) was similarly deprotected as described above using concentrated H2SO4 in EtOAc to give 100% N-methyl-4-fluoro-L-phenylalanyl-N-methyl-L-valyl-3-tert-butyl-L-tyrosine.

Updated Search

09890219

IT 287210-10-0P
RL: IMF (Industrial manufacture); RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation tripeptide derivs. by sequential coupling of N-methyl-L-valine derivs. and L-phenylalanine derivs. to 3-tert-butyl-L-tyrosinamide derivs.)
RN 287210-10-0 HCAPLUS
CN L-Tyrosinamide, N-[(1,1-dimethylethoxy)carbonyl]-4-fluoro-N-methyl-L-phenylalanyl-N-methyl-L-valyl-3-(1,1-dimethylethyl)-N-ethyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 7 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2000:657767 HCAPLUS
DOCUMENT NUMBER: 133:249332
TITLE: Method for selecting peptides capable of binding with glycolipid
INVENTOR(S): Sato, Tomonori; Okahata, Yoshio; Ishikawa, Hiroshi; Ogino, Koichi; Taki, Takao
PATENT ASSIGNEE(S): Ohtsuka Pharmaceutical Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 21 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2000253900	A	20000919	JP 2000-460	20000105
PRIORITY APPLN. INFO.:			JP 1999-769 A	19990105

AB A method is provided for specifically selecting peptides capable of binding with a glycolipid (e.g., ganglioside) by a phage display method. In this method, the biopanning with the phage-displayed peptide library is performed using ganglioside monolayer, and the binding of phage to ganglioside monolayer is monitored by a quartz-crystal microbalance.

Updated Search

09890219

Three peptides capable of binding with ganglioside GM1 are obtained by this method, and are determined to possess the amino acid sequence shown by Seq ID1-3. These peptides appear useful as a research tool to elucidate the mechanism of ganglioside function.

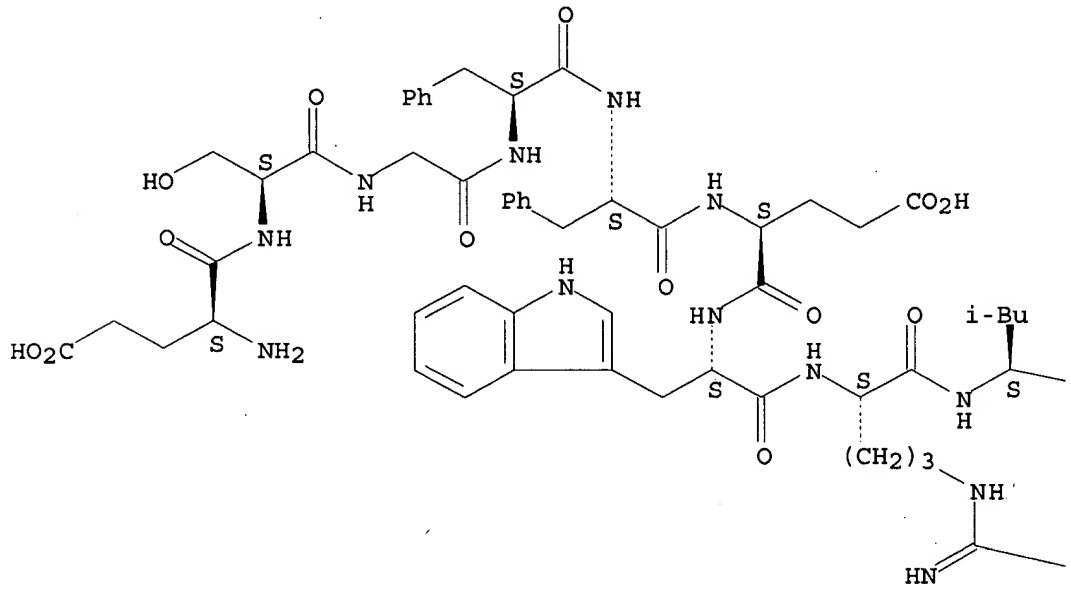
IT 294642-21-0
RL: PRP (Properties)
(unclaimed sequence; method for selecting peptides capable of binding with glycolipid)

RN 294642-21-0 HCAPLUS

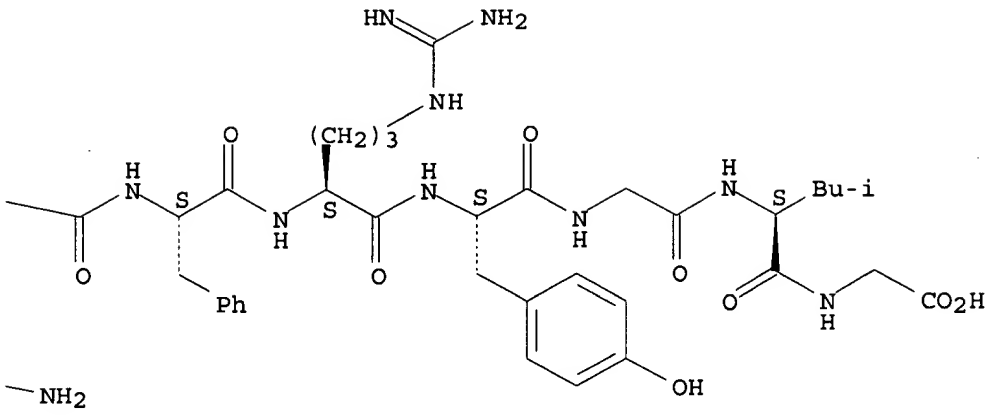
CN Glycine, L-α-glutamyl-L-serylglycyl-L-phenylalanyl-L-phenylalanyl-L-α-glutamyl-L-tryptophyl-L-arginyl-L-leucyl-L-phenylalanyl-L-arginyl-L-tyrosylglycyl-L-leucyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



Updated Search



L9 ANSWER 8 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2000:285563 HCAPLUS
DOCUMENT NUMBER: 133:84664
TITLE: Selective antagonism by naloxonazine of antinociception by Tyr-d-Arg-Phe-β-Ala, a novel dermorphin analogue with high affinity at μ-opioid receptors
AUTHOR(S): Sakurada, S.; Takeda, S.; Sato, T.; Hayashi, T.; Yuki, M.; Kutsuwa, M.; Tan-No, K.; Sakurada, C.; Kisara, K.; Sakurada, T.
CORPORATE SOURCE: Department of Physiology and Anatomy, Tohoku Pharmaceutical University, Aoba-ku, Sendai, Japan
SOURCE: European Journal of Pharmacology (2000), 395(2), 107-112
CODEN: EJPHAZ; ISSN: 0014-2999
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB To examine the role of μ-opioid receptor subtypes, we assessed the antinociceptive effect of H-Tyr-d-Arg-Phe-β-Ala-OH (TAPA), an analog of dermorphin N-terminal peptide in mice, using the tail-flick test. Intracerebroventricularly (i.c.v.) or intrathecally (i.t.) injected TAPA produced potent antinociception with tail-flick as a thermal noxious stimulus. The selective μ1-opioid receptor antagonist, naloxonazine (35 mg/kg, s.c.), or the selective μ-opioid receptor antagonist, β-funaltrexamine, 24 h before testing antagonized the antinociceptive effect of i.t. or i.c.v. TAPA on the response to noxious stimuli. Pretreatment with β-funaltrexamine completely antagonized the antinociception by both i.c.v. and i.t. administered TAPA and [d-Ala2, Me-Phe4, Gly(ol)5]enkephalin (DAMGO). Especially in the tail-flick test, pretreatment with naloxonazine produced a marked rightward displacement of the i.t. TAPA dose-response curve for antinociception. Though DAMGO is a highly selective μ-opioid receptor agonist, pretreatment with naloxonazine partially blocked the antinociceptive response to DAMGO after

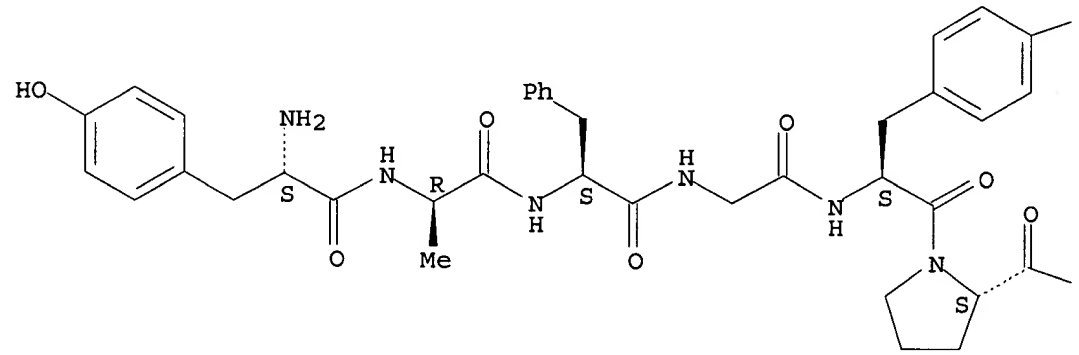
i.c.v., but not after i.t. injection. These results indicate that TAPA can act as a highly selective μ1-opioid receptor agonist (notable naloxonazine-sensitive receptor agonist) at not only the supraspinal level, but also the spinal level. These data also reveal different antinociceptive mechanisms for DAMGO and for TAPA.

IT 77614-16-5, Dermorphin
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(selective antagonism by naloxonazine of antinociception by Tyr-d-Arg-Phe-β-Ala, a novel dermorphin analog with high affinity at μ-opioid receptors)

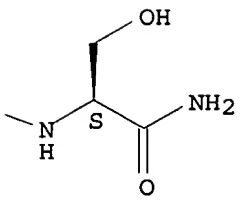
RN 77614-16-5 HCAPLUS
CN Dermorphin (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 9 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1999:139868 HCAPLUS
DOCUMENT NUMBER: 130:196958
TITLE: Preparation of 3-tert-butyl-L-tyrosinamide-containing

Updated Search

09890219

peptides and related compounds exhibiting a motilin receptor antagonism

INVENTOR(S): Kotake, Ken-ichiro; Kozono, Toshiro; Sato, Tsutomu; Takanashi, Hisanori

PATENT ASSIGNEE(S): Chugai Seiyaku Kabushiki Kaisha, Japan

SOURCE: PCT Int. Appl., 144 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: Japanese

FAMILY ACC. NUM. COUNT: 1

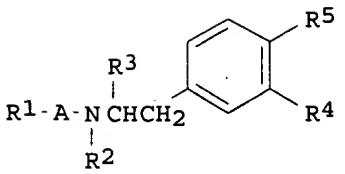
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 9909053	A1	19990225	WO 1998-JP3627		19980814
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG					
TW 460478	B	20011021	TW 1998-87113211		19980811
CA 2301687	A1	19990225	CA 1998-2301687		19980814
AU 9886490	A	19990308	AU 1998-86490		19980814
AU 741216	B2	20011129			
JP 2000044595	A	20000215	JP 1998-229586		19980814
JP 3583928	B2	20041104			
EP 1006122	A1	20000607	EP 1998-937826		19980814
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI					
US 6255285	B1	20010703	US 2000-485620		20000215
PRIORITY APPLN. INFO.:			JP 1997-255879	A	19970815
			JP 1998-186802	A	19980528
			WO 1998-JP3627	W	19980814

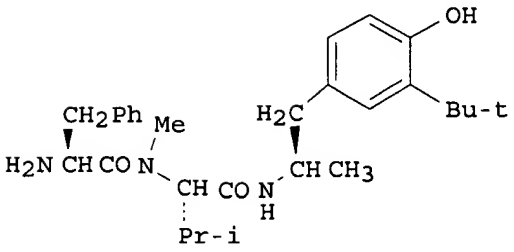
OTHER SOURCE(S): MARPAT 130:196958

GI

Updated Search



I



II

AB Phenethylamine derivs. represented by general formula [I; wherein A represents an amino acid or α-substituted amino acid residue; R1 represents R6CO, (un)substituted C2-7 linear or branched alkyl, C3-8 alkenyl, or C3-8 alkynyl; R2 represents hydrogen, C1-3 linear or branched alkyl; R3 represents COR7, (un)substituted C1-5 linear or branched alkyl, C2-5 alkenyl, or C2-5 alkynyl; R4 represents H, C1-6 linear or branched alkyl, C2-6 alkenyl, C2-6 alkynyl, etc.; R5 represents hydroxy or C1-4 n-alkoxy; R6 represents (un)substituted C1-6 linear or branched alkyl, C2-7 alkenyl, or C2-7 alkynyl, optionally benzene- or heterocyclic ring-condensed C3-7 cycloalkyl, (un)substituted C6-12 aromatic ring, (un)substituted C3-12 (un)saturated heterocyclic ring, (un)substituted NH2, (un)substituted linear or branched C1-5 alkoxy, C2-6 alkenyloxy, C2-6 alkynyloxy, etc.; and R7 represents H, (un)substituted C1-5 linear or branched alkyl, C3-7 cycloalkyl, (un)substituted NH2, OH, linear or branched alkyl C1-6 alkoxy, or C3-7 cycloalkyloxy] are prepared Also claimed are a motilin receptor antagonist, an inhibitor of digestive tract motility, and a remedy for high blood motilin. They are also useful for the treatment of irritable bowel syndrome. Thus, Nα-methyl-N-[2-(3-tert-butyl-4-hydroxyphenyl)-1-methylethyl]-L-valinamide was condensed with Boc-Phe-OH using HOBT and DIC in DMF at room temperature for 2.5 days followed by deprotection with CF3CO2H in CH2Cl2 to give the title compound (II). II in vitro showed IC50 of 1.9 nM for inhibiting the binding of [125I]motilin motilin receptor preparation from rabbit ileum mucous membrane.

IT 220806-34-8P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of 3-tert-butyl-L-tyrosinamide-containing peptide compds. as motilin receptor antagonists, inhibitors of digestive tract motility, and remedy for high blood motilin)

RN 220806-34-8 HCAPLUS

CN L-Tyrosinamide, L-phenylalanyl-(2S)-2-phenylglycyl-3-(1,1-dimethylethyl)-, mono(trifluoroacetate) (salt) (9CI) (CA INDEX NAME)

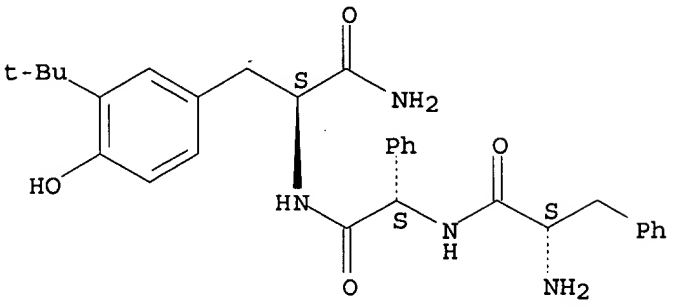
Updated Search

09890219

CM 1

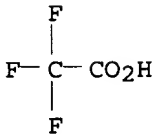
CRN 220806-33-7
CMF C30 H36 N4 O4

Absolute stereochemistry.



CM 2

CRN 76-05-1
CMF C2 H F3 O2



REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 10 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1998:749277 HCAPLUS
DOCUMENT NUMBER: 130:163052
TITLE: Comparison of opioid activity between a N-terminal tetrapeptide analog of dermorphin, H-Tyr-D-Arg-Phe-β-Ala-OH and morphine
AUTHOR(S): Sato, Takumi; Takahashi, Norio; Tan-No, Koichi; Kisara, Kensuke; Sakurada, Tsukasa; Sakurada, Shinobu
CORPORATE SOURCE: Departments of Pharmaceutics, Tohoku College of Pharmacy, Sendai, Japan
SOURCE: Methods and Findings in Experimental and Clinical Pharmacology (1998), 20(7), 581-586
CODEN: MFEPDX; ISSN: 0379-0355
PUBLISHER: Prous Science
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The antinociceptive properties of dermorphin tetrapeptide analog, H-Tyr-D-Arg-Phe-β-Ala-OH (TDAPA) were compared with morphine in mice. In the tail-pressure test, s.c. injected TDAPA and morphine produced significant antinociceptive activity. Pretreatment with naloxonazine (35

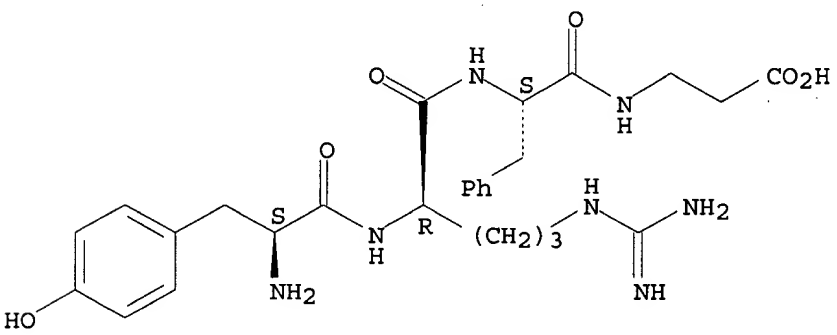
Updated Search

mg/kg, s.c., 24 h before testing) significantly antagonized the activity induced by TDAPA, but not morphine. The ED50 values of TDAPA changed from 0.39 mg/kg to 1.7 mg/kg by naloxonazine pretreatment. In the formalin test, both TDAPA and morphine exhibited dose-related antinociceptive activity with ED50 values of 0.49 mg/kg and 2.5 mg/kg, resp. Both drug activities were significantly antagonized by naloxonazine. These results indicate different mechanisms of action for TDAPA and morphine, suggesting TDAPA is highly selective for the μ1-opioid receptor and may be clin. useful.

IT 104347-80-0
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(comparison of opioid activity between N-terminal tetrapeptide analog of dermorphin, H-Tyr-D-Arg-Phe-β-Ala-OH and morphine)

RN 104347-80-0 HCAPLUS
CN β-Alanine, L-tyrosyl-D-arginyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 11 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1998:668227 HCAPLUS
DOCUMENT NUMBER: 130:20517
TITLE: Differential antagonistic effect of intracerebroventricularly and intrathecally administered β-funaltrexamine on dermorphin analog- and morphine-induced antinociception in mice
AUTHOR(S): Takahashi, Norio; Sakurada, Shinobu; Yonezawa, Akihiko; Arai, Kinue; Sato, Takumi; Kawamura, Shunsuke; Niijima, Fukie; Kisara, Kensuke
CORPORATE SOURCE: Tohoku Coll. Pharm., Sendai, 981-8558, Japan
SOURCE: Annual Report of the Tohoku College of Pharmacy (1997), 44, 241-251
CODEN: TYKNAQ; ISSN: 0495-7342
PUBLISHER: Tohoku Yakka Daigaku
DOCUMENT TYPE: Journal
LANGUAGE: Japanese

AB The authors investigated the antagonistic effects of β-funaltrexamine (β-FNA) on the antinociception induced by [Tyr-D-Arg-Phe-β-Ala-OH:TAPA] and morphine in mice. Pretreatment with β-FNA given intracerebroventricularly (i.c.v.) and intrathecally (i.t.) shifted dose-response curve of TAPA 6.76-fold and 4.71-fold to the right, resp.

Updated Search

09890219

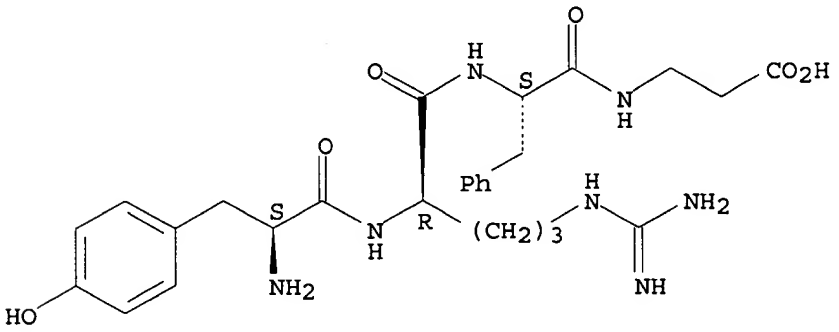
Pretreatment with β-FNA given i.c.v. and i.t. shifted dose-response curve of morphine 23.75-fold and 2.26-fold to the right, resp. Antinociception of s.c. administered morphine was more potently blocked by pretreatment with i.c.v. β-FNA than by the i.t. route. TAPA-induced antinociception was similarly blocked by either i.c.v. or i.t. β-FNA. These results suggest that s.c. morphine-induced antinociception is largely mediated through supraspinal antinociceptive systems, whereas potent antinociception of s.c. TAPA is contributed by not only supraspinal level but also spinal level.

IT 104347-80-0
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(differential antagonistic effect of intracerebroventricularly and intrathecally administered β-funaltrexamine on dermorphin analog- and morphine-induced antinociception)

RN 104347-80-0 HCAPLUS

CN β-Alanine, L-tyrosyl-D-arginyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L9 ANSWER 12 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1998:112246 HCAPLUS

DOCUMENT NUMBER: 128:176178

TITLE: Compounds that inhibit interaction between signal-transducing proteins and the GLGF (PDX/DHR) domain and uses thereof

INVENTOR(S): Sato, Taka-aki; Yanagisawa, Junn

PATENT ASSIGNEE(S): Trustees of Columbia University In the City of New York, USA

SOURCE: PCT Int. Appl., 108 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9805347	A1	19980212	WO 1997-US12677	19970718
W: AU, CA, JP, MX, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 2002058607	A1	20020516	US 1996-681219	19960722
US 6911526	B2	20050628		
CA 2260742	A1	19980212	CA 1997-2260742	19970718

Updated Search

09890219

AU 9740424	A	19980225	AU 1997-40424		19970718
AU 735287	B2	20010705			
EP 935467	A1	19990818	EP 1997-937999		19970718
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI					
CN 1230120	A	19990929	CN 1997-197769		19970718
JP 2001505540	T	20010424	JP 1998-507935		19970718
KR 2000029469	A	20000525	KR 1999-700466		19990121
US 2003203414	A1	20031030	US 1999-230111		19990517
PRIORITY APPLN. INFO.:			US 1996-681219	A2	19960722
			WO 1997-US12677	W	19970718

AB This invention provides for a composition capable of inhibiting specific binding between a signal-transducing protein and a cytoplasmic protein. This invention also provides a method of identifying a compound capable of inhibiting specific binding between a signal-transducing protein and a cytoplasmic protein. This invention also provides a method of inhibiting the proliferation of cancer cells. This invention also provides a method of treating cancer with a composition in an amount effective to result in an amount in apoptosis of the cells. This invention also provides a method of inhibiting the proliferation of virally infected cells. This invention also provides for a method of treating a virally-infected subject with a composition in an amount effective to result in apoptosis of the cells. This invention also provides for pharmaceutical compns.

IT 203438-19-1P
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PNU (Preparation, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); PROC (Process); USES (Uses)
(compds. that inhibit interaction between signal-transducing proteins and the GLGF (PDX/DHR) domain and uses thereof)

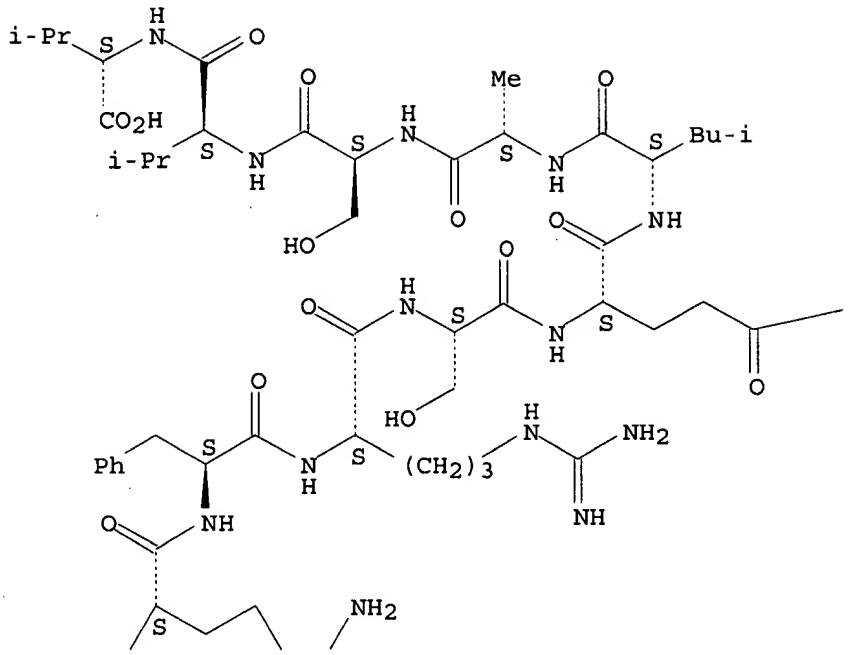
RN 203438-19-1 HCAPLUS

CN L-Valine, L-α-aspartyl-L-seryl-L-α-glutamyl-L-methionyl-L-tyrosyl-L-glutaminyl-L-phenylalanyl-L-arginyl-L-seryl-L-glutaminyl-L-leucyl-L-alanyl-L-seryl-L-valyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

Updated Search

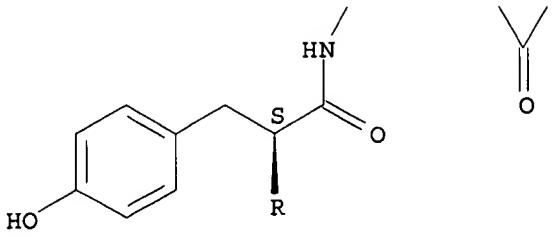
PAGE 1-A

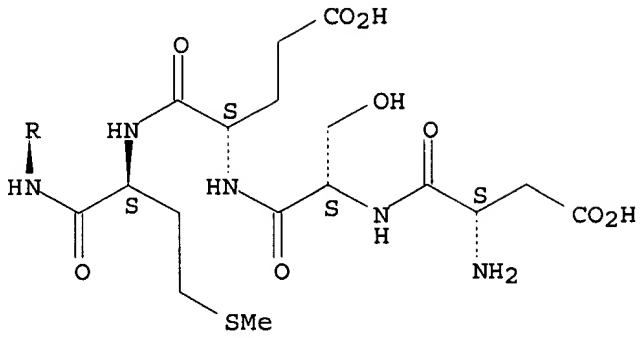


PAGE 1-B

—NH_2

Updated Search





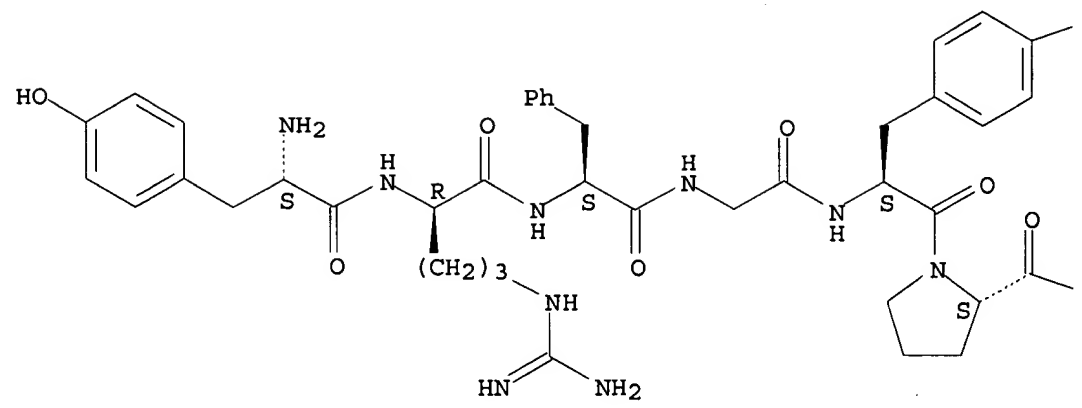
REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 13 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1994:646501 HCAPLUS
DOCUMENT NUMBER: 121:246501
TITLE: Potent opioid activities of (D-Arg2) dermorphin analogs
AUTHOR(S): Sakurada, Shinobu; Sato, Takumi; Kisara, Kensuke
CORPORATE SOURCE: Tohoku Coll. Pharm., Sendai, 981, Japan
SOURCE: Annual Report of the Tohoku College of Pharmacy (1993), 40, 1-19
CODEN: TYKNAQ; ISSN: 0495-7342
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
AB Review, with 53 refs. A correlation between dermorphin structure and opioid activities was discussed based on a variety of analogs of (D-Arg2) dermorphin. The minimal structure of opioid activities was the tripeptide of N-terminus for (D-Arg2) dermorphin, although it was the tetrapeptide for dermorphin. Genetic anal. for dermorphin showed that D-Ala was replaced by a post-transformation process.
IT 96425-96-6D, (D-Arg2) dermorphin, analogs
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(opioid activities of dermorphin analogs)
RN 96425-96-6 HCAPLUS
CN Dermorphin, 2-D-arginine- (9CI) (CA INDEX NAME)

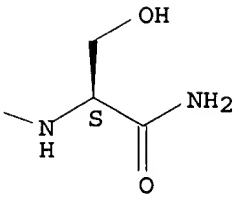
Absolute stereochemistry.

Updated Search

PAGE 1-A



PAGE 1-B



L9 ANSWER 14 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1992:228388 HCAPLUS
DOCUMENT NUMBER: 116:228388
TITLE: Comparison of opioid properties between D-Arg-containing dipeptides and tetrapeptides
AUTHOR(S): Sato, Takumi; Sakurada, Shinobu; Sakurada, Tsukasa; Kisara, Kensuke; Suzuki, Kenji
CORPORATE SOURCE: Dep. Pharm., Tohoku Coll. Pharm., Sendai, 981, Japan
SOURCE: Biochemical Pharmacology (1992), 43(4), 717-23
CODEN: BCPCA6; ISSN: 0006-2952
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Since the D-Arg-containing dipeptides, H-Tyr-D-Arg-OMe (TDA) and H-Tyr(Et)-D-Arg-OMe, and the D-Arg2-substituted dermorphin analogs, H-Tyr-D-Arg-Phe-Gly-OTe (TDAPG) and H-Tyr(Et)-D-Arg-Phe-Gly-OEt, gave different pharmacol. responses in vivo, opioid interaction and structure-activity relations have been investigated in vitro. In the isolated guinea pig ileum assay, the tetrapeptides were potently inhibitory, their activity markedly exceeding that of the dipeptides. In particular, the first tetrapeptide had twice the activity of morphine,

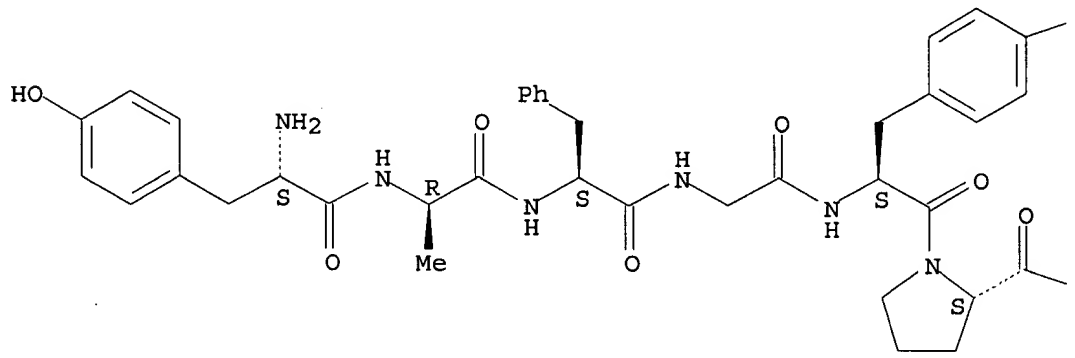
whereas the potencies of the dipeptides were less than 5% that of morphine. Also, in the opioid receptor binding assay, tetrapeptides had a higher affinity than did the dipeptides. IC50 values of tetrapeptides were 8.46 and 23.7 nM, resp., which were lower than that of morphine. Ethylation of the Tyr residue of TDA much increased the opioid activity, whereas similar modification of TDAPG greatly decreased opioid activity. All peptides used were extremely stable to aminopeptidase-M and carboxypeptidase-Y and had an inhibitory effect on enkephalin (EK)-degrading enzymes. Apparently, the effects of the tetrapeptides are due mainly to specific interaction with opioid receptors, whereas the dipeptides do not act specifically on the opioid receptors, but are involved in non-opioid mechanisms. The resistance to enzymes and inhibitory effect of the peptides used on the EK-degrading enzymes may also account for their potent and long-lasting opioid-like activities.

IT 77614-16-5, Dermorphin
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(opioid activity of, structure in relation to)

RN 77614-16-5 HCAPLUS
CN Dermorphin (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



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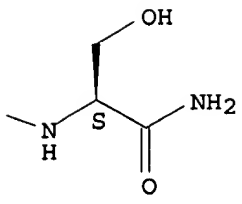


Updated Search

09890219

L9 ANSWER 15 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1990:196672 HCAPLUS
DOCUMENT NUMBER: 112:196672
TITLE: Neutrophil-activating factors and its manufacture with serum-independent human cells
INVENTOR(S): Shionoya, Hiroshi; Koyanagi, Nozomi; Sato, Toshitaka; Kuwata, Manabu; Koide, Jun; Miyoshi, Isao
PATENT ASSIGNEE(S): Eisai Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 11 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 01161000	A	19890623	JP 1987-318328	19871216

PRIORITY APPLN. INFO.: JP 1987-318328 19871216

AB Antitumor and antibacterial neutrophil-activating factor (NAS) having a mol. weight of 25,000 and a partially defined amino acid sequence is manufactured by cultivating serum-independent strain A-6 of cell line MT-2 that is established from human umbilical leukocytes. Thus, strain A-6 isolated from cell line MT-2 was cultivated in the serum-free RPMI-1640 medium. The medium was subsequently condensed, chromatographed, gel-filtered in presence of 6 M urea, and purified by the reverse-phased HPLC to obtain NAS. The effect of NAS on antitumor activity of neutrophiles against mast cell tumor P815 was demonstrated.

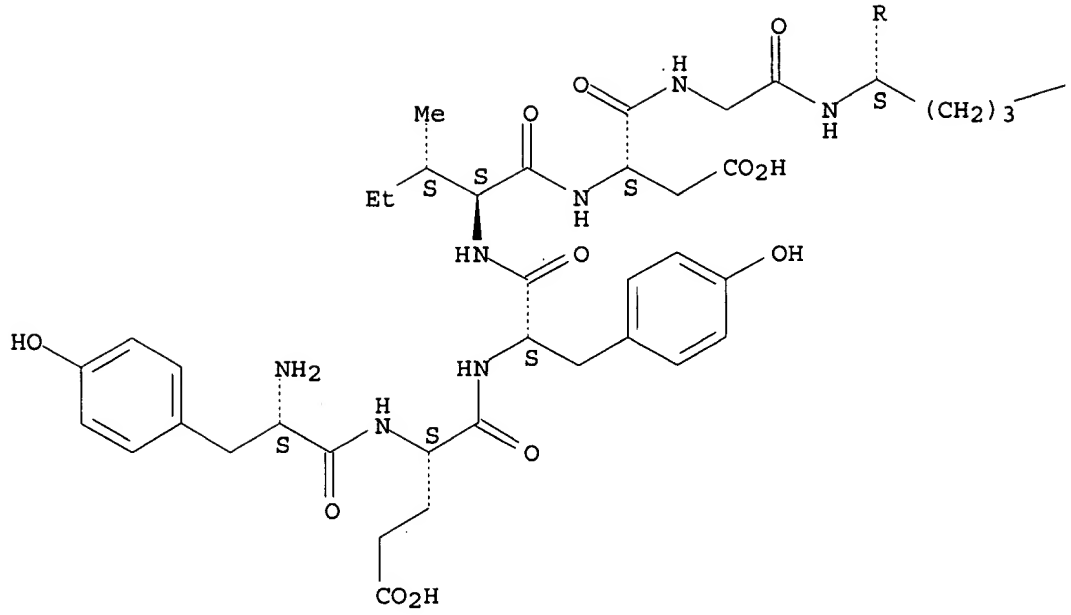
IT 126738-60-1
RL: BIOL (Biological study)
(amino acid sequence in neutrophil-activating factor)

RN 126738-60-1 HCAPLUS
CN L-Serine, N-[N2-[N-[N-[N-[N-(N-L-tyrosyl-L-α-glutamyl)-L-tyrosyl]-L-isoleucyl]-L-α-aspartyl]glycyl]-L-arginyl]- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

Updated Search

PAGE 1-A



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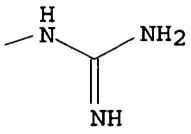
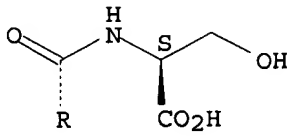


PAGE 2-A



L9 ANSWER 16 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1989:166399 HCAPLUS

DOCUMENT NUMBER: 110:166399

TITLE: A new class opioid peptide, [D-Arg2,β-Ala4]-dermorphin tetrapeptide; physical dependence liability in mice

AUTHOR(S): Chaki, K.; Sakurada, S.; Sakurada, T.; Sato, T.; Kawamura, S.; Kisara, K.; Sasaki, Y.; Suzuki, K.

CORPORATE SOURCE: Dep. Pharmacol., Tohoku Coll. Pharm., Sendai, 981, Japan

Updated Search

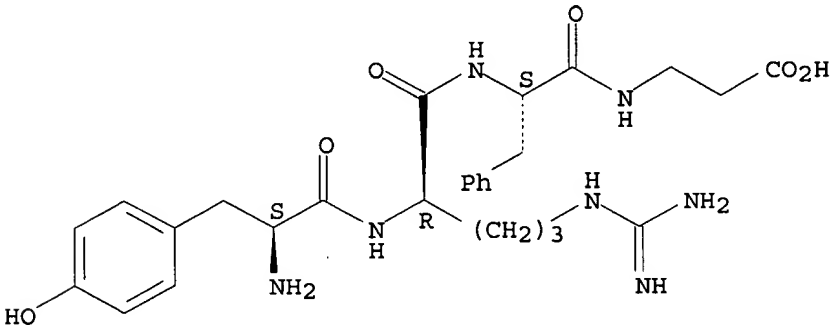
09890219

SOURCE: Neuropeptides (Edinburgh, United Kingdom) (1989), 13(2), 83-8
CODEN: NRPPDD; ISSN: 0143-4179
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Development of morphine-like phys. dependence on [D-Arg2,β-Ala4]-dermorphin tetrapeptide (H-Tyr-D-Arg-Phe-β-Ala-OH) was evaluated and compared with the phys. dependence liability of morphine or pentazocine. The degree of phys. dependence was assessed by naloxone-precipitated jumping behavior in mice after treatment with a single dose of each compound The number of jumps and the time of latency to first jump were recorded in this experiment The number of jumps in a group pretreated with the peptide was less than that in the morphine-treated group. In addition, latency to the appearance of the first jump in the peptide-treated mice was later than that in the morphine-treated group. Thus, the phys. dependence induced by [D-Arg2,β-Ala4]-dermorphin tetrapeptide may be less marked than that produced by morphine. It is also interesting to note that the antinociceptive effect of this opioid peptide is more powerful and of longer duration than that induced by morphine or pentazocine.

IT 104347-80-0
RL: BIOL (Biological study)
(antinociceptive activity and dependence liability of)
RN 104347-80-0 HCAPLUS
CN β-Alanine, L-tyrosyl-D-arginyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L9 ANSWER 17 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1989:108356 HCAPLUS
DOCUMENT NUMBER: 110:108356
TITLE: Comparison of the antinociceptive effects of new [D-Arg2]-dermorphin tetrapeptide analogs and morphine in mice
AUTHOR(S): Chaki, Kyoji; Sakurada, Shinobu; Sakurada, Tsukasa; Sato, Takumi; Kawamura, Shunsuke; Kisara, Kensuke; Watanabe, Hiromi; Suzuki, Kenji
CORPORATE SOURCE: Dep. Pharmacol., Tohoku Coll. Pharm., Sendai, 981, Japan
SOURCE: Pharmacology, Biochemistry and Behavior (1988), 31(2), 439-44
CODEN: PBBHAU; ISSN: 0091-3057
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The antinociceptive effects of synthetic dermorphin tetrapeptide analogs

Updated Search

containing D-Arg in position 2, H-Tyr-D-Arg-Phe-Gly-NH2 and H-Tyr-D-Arg-Phe-β-Ala-OH, were measured in mice by the tail-pressure test. The antinociceptive effect produced by intracerebroventricular (ICV), intrathecal (IT), and s.c. administration of either peptide was greater than that produced by morphine. Oral administration of the peptides showed approx. the same antinociceptive potency as morphine. In addition, the antinociceptive effect produced by s.c. or oral administration of either peptide was of longer duration than morphine. Pretreatment with naloxone resulted in early complete antagonism of the antinociceptive effects produced by ICV and IT administration of either peptide or morphine. Dose ratios (ICV/IT) or H-Tyr-D-Arg-Phe-Gly-NH2 and H-Tyr-D-Arg-Phe-β-Ala-OH, which were calculated from the AD50 (Antinociceptive Dose = 50% maximal possible effect) values, were 5.8 and 6.2, resp., whereas that of morphine was only 1.46. Thus, the mechanisms of the antinociceptive effects of [D-Arg2]-dermorphin tetrapeptide analogs apparently differ from those of morphine, and these peptides may possess higher affinities than does morphine for opioid receptors in the spinal cord.

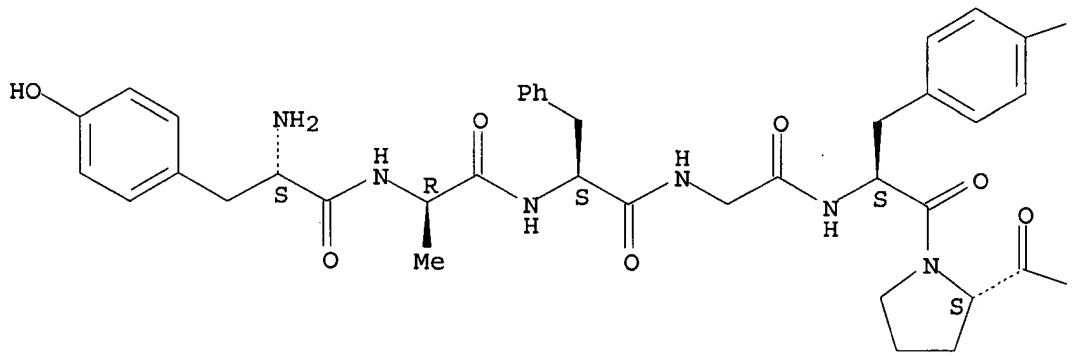
IT 77614-16-5D, Dermorphin, analogs
RL: BIOL (Biological study)
(antinociceptive activity of, administration route and structure in relation to)

RN 77614-16-5 HCAPLUS

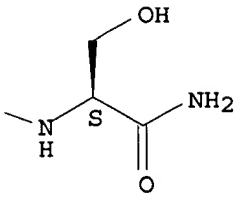
CN Dermorphin (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



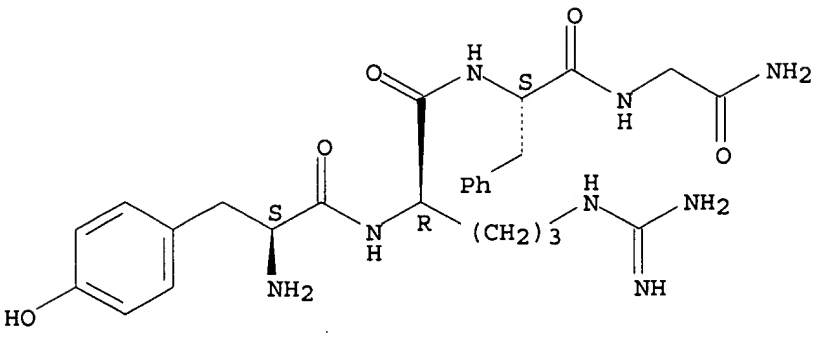
—OH



L9 ANSWER 18 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1988:563803 HCAPLUS
DOCUMENT NUMBER: 109:163803
TITLE: Antinociception and physical dependence produced by [D-Arg2]dermorphin tetrapeptide analogs and morphine in rats
AUTHOR(S): Chaki, Kyoji; Kawamura, Shunsuke; Kisara, Kensuke; Sakurada, Shinobu; Sakurada, Tsukasa; Sasaki, Yusuke; Sato, Takumi; Susuki, Kenji
CORPORATE SOURCE: Dep. Pharmacol. Biochem., Tohoku Coll. Pharm., Sendai, 981, Japan
SOURCE: British Journal of Pharmacology (1988), 95(1), 15-22
CODEN: BJPCBM; ISSN: 0007-1188
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The antinociceptive effects of [D-Arg2]-dermorphin tetrapeptide analogs, H-Tyr-D-Arg-Phe-Gly-NH2 and H-Tyr-D-Arg-Phe-β-Ala-OH, when administered s.c. in rats were measured by the tail-flick test. In addition, the appearance of typical withdrawal signs upon cessation of administration or on subsequent treatment with naloxone were measured after chronic administration of either peptide or of morphine. The dose of peptides and of morphine in the phys. dependence test was determined from the AD50 (dose with 50% of maximal antinociceptive effect) to inhibit the tail-flick test in rats. Doses of 4-64-fold the AD50 doses were employed in the s.c. administration schedules. The intensity of the antinociception induced by either peptide was greater than that produced by morphine. Moreover, the antinociception induced by the peptides was of much longer duration than that produced by morphine. Abrupt withdrawal after chronic administration of either peptide produced only slight loss of body weight In contrast, morphine withdrawal produced sharp loss of body weight Naloxone-precipitated withdrawal signs after chronic administration of either peptide were less intense than those after chronic morphine. Thus, the antinociception produced by these peptides is more intense and of longer duration that that produced by morphine, and the phys. dependence produced by these peptides is less marked than that produced by morphine.
IT 100304-60-7
RL: BIOL (Biological study)
(analgesia and phys. dependence production by, morphine in relation to)
RN 100304-60-7 HCAPLUS
CN Glycinamide, L-tyrosyl-D-arginyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

09890219

Absolute stereochemistry.



L9 ANSWER 19 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1988:16814 HCAPLUS
DOCUMENT NUMBER: 108:16814
TITLE: Production and characterization of monoclonal antibodies against amino-terminus of human α-atrial natriuretic polypeptide
AUTHOR(S): Naomi, Shojiro; Umeda, Teruhisa; Sato, Tatsuo ; Harada, Nobuyuki; Tominaga, Akira; Takatsu, Kiyoshi
CORPORATE SOURCE: Med. Sch., Kumamoto Univ., Kumamoto, 860, Japan
SOURCE: Hybridoma (1987), 6(4), 433-40
CODEN: HYBRDY; ISSN: 0272-457X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Monoclonal antibodies directed against human α-atrial natriuretic polypeptide (α-ANP; human, 1-28) were obtained by somatic cell fusion between P3-X63-Ag8.653 myeloma cells and spleen cells from a BALB/c mouse immunized with human α-ANP selectively coupled to keyhole limpet hemocyanin. From the anal. of polyclonal sera with respect to determinant specificity before the fusion, the strategy was primarily used to pick up monoclonal antibody specific for the N-terminal residues of human α-ANP. Screenings of antibodies in the hybridoma culture supernatants were performed by binding to iodinated synthetic human α-ANP. Two stable clones producing anti-human α-ANP antibodies, designated 13A1 and 10B1, were obtained by the limiting dilution technique. The ability of ANP (rat, 1-28) to inhibit binding of 125I-labeled human α-ANP to these antibodies was almost equipotent to ANP (human, 1-28). However, ANP fragments (human, 7-28) and (18-28) did not inhibit the binding completely. Apparently both 13A1 and 10B1 monoclonal antibodies can specifically recognize the N-terminus of human α-ANP, and may be useful tools to investigate receptor binding of human α-ANP by the antagonizing effect.
IT 88898-17-3, Rat atrial natriuretic peptide 1-28
RL: BIOL (Biological study)
(atriopeptin monoclonal antibodies reaction with)
RN 88898-17-3 HCAPLUS
CN Atrial natriuretic peptide-28 (rat) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L9 ANSWER 20 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1987:591140 HCAPLUS
DOCUMENT NUMBER: 107:191140

Updated Search

09890219

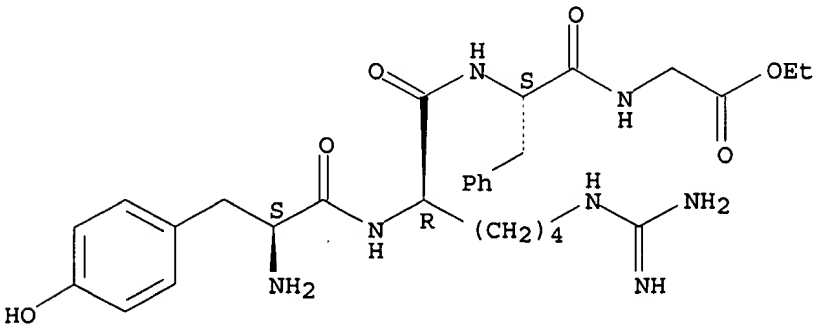
TITLE: Opioid activities of D-Arg2-substituted tetrapeptides
AUTHOR(S): Sato, Takumi; Sakurada, Shinobu; Sakurada, Tsukasa; Furuta, Seiichi; Chaki, Kyoji; Kisara, Kensuke; Sasaki, Yusuke; Suzuki, Kenji
CORPORATE SOURCE: Dep. Pharmacol., Tohoku Coll. Pharm., Sendai, 983, Japan
SOURCE: Journal of Pharmacology and Experimental Therapeutics (1987), 242(2), 654-9
CODEN: JPETAB; ISSN: 0022-3565
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The antinociceptive effects and mechanisms of action of H-Tyr-D-Ala-Phe-Gly-OH, H-Tyr-D-Arg-Phe-Gly-OH, and H-Tyr-D-Arg-Phe-sarcosine(Sar)-OH were investigated. The ED50 values of these peptides were 510.0, 8.2, and 2.0 pmol, resp., when administered intracerebroventricularly in the mouse tail-pressure test (dermorphin = 5.7 pmol and morphine = 1.2 nmol). These activities were remarkably potent and relatively long lasting. Their IC50 values were 676.8, 23.1, and 6.6 nM, resp. (dermorphin = 3.75 and morphine = 214.3 nM) in the guinea pig isolated ileum assay, and 138.50, 5.25, and 1.10 nM, resp. (dermorphin = 3.80 and morphine = 28.00 nM) in the radioreceptor assay utilizing [3H]naloxone as the opioid receptor ligand. In the evaluation of their inhibitory effects to enkephalin-degrading enzymes, the IC50 values of H-Tyr-D-Arg-Phe-Gly-OH, H-Tyr-D-Arg-Phe-Sar-OH, and H-Tyr-D-Ala-Phe-Gly-OH were 5.4, 14.5, and >50.0 μM, resp. (bestatin = 0.1 μM) against aminopeptidase and 1.18, 1.40, >50.0 μM, resp. (captopril = 0.38 and D-Phe-2S-,3R-3-amino-2-hydroxy-4-phenylbutanoic acid = >100 μM) against the cleaving enzymes of enkephalin at its Gly3-Phe4 bond. Evidently, the marked antinociceptive potency of H-Tyr-D-Arg-Phe-Gly-OH and H-Tyr-D-Arg-Phe-Sar-OH is mainly due to high opioid receptor affinity. Their inhibitory effects on enkephalin-degrading enzymes and enzymic stability also greatly contribute to their potent and long-lasting opioid activities. Structure-activity relations of the tetrapeptides are discussed.

IT 96425-94-4
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(analgesic activity of)

RN 96425-94-4 HCAPLUS
CN Glycine, N-[N-[N6-(aminoiminomethyl)-N2-L-tyrosyl-D-lysyl]-L-phenylalanyl]-, ethyl ester (9CI) (CA INDEX NAME)

Absolute stereochemistry.



Updated Search

09890219

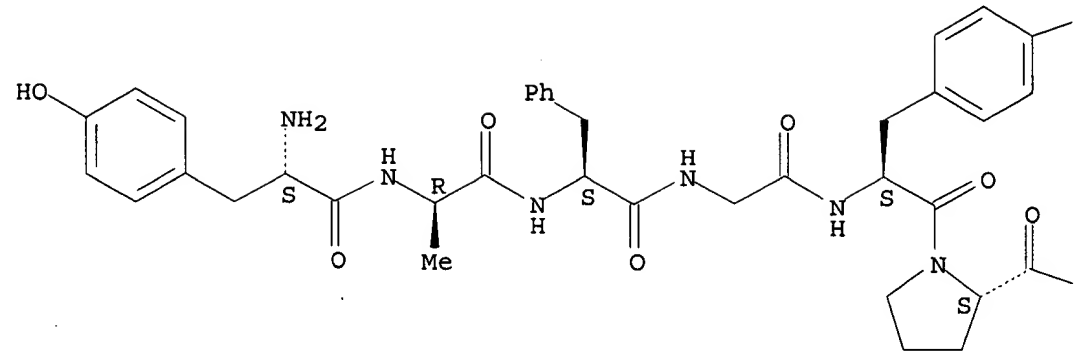
L9 ANSWER 21 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1986:102638 HCAPLUS
DOCUMENT NUMBER: 104:102638
ORIGINAL REFERENCE NO.: 104:16102h,16103a
TITLE: Dermorphin analogs containing D-kyotorphin: structure-antinociceptive relationships in mice
AUTHOR(S): Kisara, Kensuke; Sakurada, Shinobu; Sakurada, Tsukasa; Sasaki, Yusuke; Sato, Takumi; Suzuki, Kenji; Watanabe, Hiromi
CORPORATE SOURCE: Dep. Pharmacol., Tohoku Coll. Pharm., Sendai, 983, Japan
SOURCE: British Journal of Pharmacology (1986), 87(1), 183-9
CODEN: BJPCBM; ISSN: 0007-1188
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The antinociceptive effects of synthetic dermorphin [77614-16-5] and its analogs containing D-arginine (D-Arg) in position 2 injected into the lateral cerebroventricle were examined in conscious mice. Intracerebroventricular (i.c.v.) administration of dermorphin and [D-Arg2]-dermorphin [96425-96-6] produced potent and long-lasting antinociceptive activity as assayed by the tail-pressure test. Dermorphin and [D-Arg2]-dermorphin were 210- and 52-fold more potent than morphine, resp. The antinociceptive effects produced by these heptapeptides were antagonized by a low dose (0.5 mg/kg, i.p.) of the opioid antagonist naloxone. The concentration levels for half-maximal antinociception for [D-Arg2]-dermorphin-(1-6) [100304-61-8], -(1-5) [100304-62-9], and -(1-4) [100304-60-7] were different from that for [D-Arg2]-dermorphin. The shortest fragment, [D-Arg2]-dermorphin-(1-2) [100304-63-0], had little activity, whereas [D-Arg2]-dermorphin-(1-3) [83934-32-1] exhibited activity and was 10-fold more potent than morphine. [D-Arg2]-dermorphin analogs showed almost identical effects when tested on the elec. induced contractions of the guinea pig isolated ileum. Evidently, the presence of the N-terminal tripeptide in the structure of [D-Arg2]-dermorphin is of crucial importance for the manifestation of the full intrinsic opioid-like antinociceptive activity of [D-Arg2]-dermorphin, which is presumably mediated through opioid receptors in the brain.
IT 77614-16-5
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(analgesic action of, mol. structure in relation to)
RN 77614-16-5 HCAPLUS
CN Dermorphin (CA INDEX NAME)

Absolute stereochemistry.

Updated Search

09890219

PAGE 1-A



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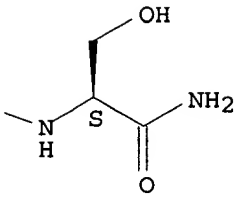


L9 ANSWER 22 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1986:34327 HCAPLUS
DOCUMENT NUMBER: 104:34327
ORIGINAL REFERENCE NO.: 104:5652h,5653a
TITLE: Studies on analgesic oligopeptides. III. Synthesis and analgesic activity after subcutaneous administration of [D-Arg2]dermorphin and its N-terminal tetrapeptide analogs
AUTHOR(S): Sasaki, Yusuke; Matsui, Michiko; Fujita, Hiroki; Hosono, Masahiro; Taguchi, Masumi; Suzuki, Kenji; Sakurada, Shinobu; Sato, Takumi; Sakurada, Tsukasa; Kisara, Kensuke
CORPORATE SOURCE: Tohoku Coll. Pharm., Sendai, 983, Japan
SOURCE: Chemical & Pharmaceutical Bulletin (1985), 33(4), 1528-36
CODEN: CPBTAL; ISSN: 0009-2363
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 104:34327
AB [D-Arg2]dermorphin (H-Tyr-D-Arg-Phe-Gly-Tyr-Pro-Ser-NH2) and 19 N-terminal tetrapeptide analogs, e.g., H-Tyr-D-Arg-Phe-Gly-OH (I), were prepared by the conventional solution method and their analgesic activities after s.c.

Updated Search

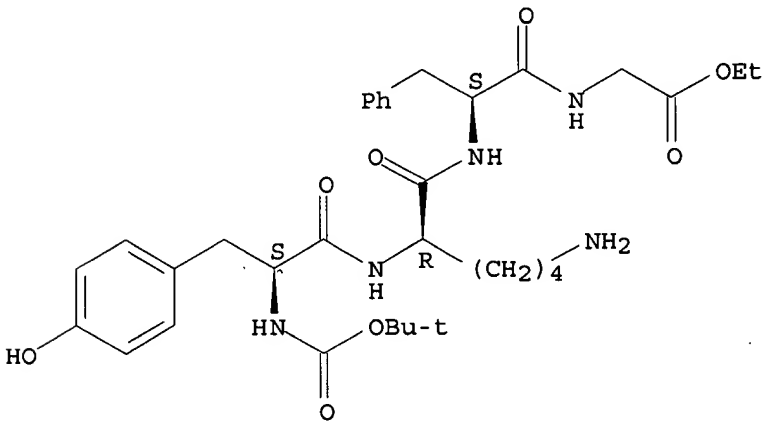
administration to mice were assessed by the tail-pressure test. [D-Arg2]dermorphin had analgesic potency equal to or slightly greater than that of dermorphin. I showed a potency 4.8 times that of morphine and comparable with that of dermorphin on a molar basis. Several analogs in which Gly4 was replaced by sarcosine or D-Ala exhibited activity greater than that of I. Replacement of Gly4 by Pro, Leu, or D-leu resulted in a marked decrease in potency, and replacement of either Phe3 by other aromatic amino acids or D-Arg2 by other basic D-amino acids gave analogs with greatly decreased activities. However, one analog whose guanidino functionality on D-Arg2 was blocked by a nitro group, showed activity one-third that of the parent peptide I. The structure-activity relationship for the tetrapeptide is discussed.

IT 99592-98-0P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation and amidination of)

RN 99592-98-0 HCAPLUS

CN Glycine, N-[N-[N2-[N-[(1,1-dimethylethoxy)carbonyl]-L-tyrosyl]-D-lysyl]-L-phenylalanyl]-, ethyl ester (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L9 ANSWER 23 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1985:400760 HCAPLUS

DOCUMENT NUMBER: 103:760

ORIGINAL REFERENCE NO.: 103:143a,146a

TITLE: A comparison of the antinociceptive and behavioral effects of D-Arg-substituted dipeptides and tetrapeptides in mice

AUTHOR(S): Sato, Takumi; Sakurada, Shinobu; Sakurada, Tsukasa; Kisara, Kensuke; Sasaki, Yusuke; Suzuki, Kenji

CORPORATE SOURCE: Dep. Pharmacol., Tohoku Coll. Pharm., Sendai, 983, Japan

SOURCE: Peptides (New York, NY, United States) (1985), 6(1), 35-40
CODEN: PPTDD5; ISSN: 0196-9781

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Intracerebroventricular administration of D-arginine (D-Arg)-substituted dipeptides, H-Tyr-D-Arg-OMe [92758-99-1] and H-Tyr(Et)-D-Arg-OMe

Updated Search

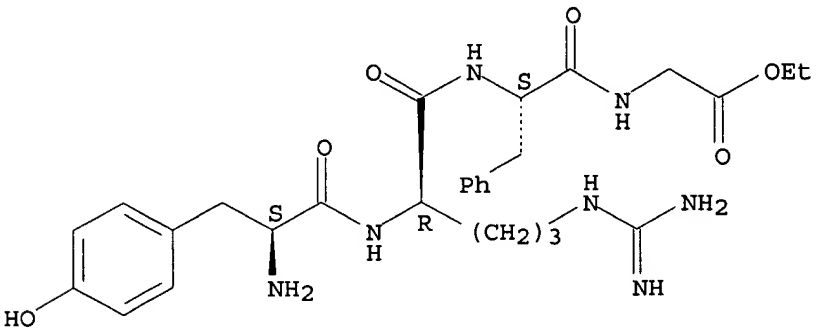
[92759-00-7] and D-Arg2-substituted N-terminal tetrapeptides of dermorphin [77614-16-5], H-Tyr-D-Arg-Phe-Gly-OEt [90549-84-1] and H-Tyr(Et)-D-Arg-Phe-Gly-OEt [92759-01-8] resulted in dose-related and naloxone-reversible antinociceptive effects. Among them, tetrapeptides not only exhibited much more potent and prolonged activities than dipeptides but also were significantly antagonized even by a low dose of naloxone. Spontaneous motor activity was lowered by dipeptides throughout the observation period, which was scarcely antagonized by naloxone. Tetrapeptides elicited locomotor hyperactivity following an initial locomotor suppression. Only the locomotor hyperactivity was significantly antagonized by naloxone. Evidently, tetrapeptides induce their effects via opioid receptors, whereas the effects of dipeptides are nonspecifically involved in various systems.

IT 90549-84-1
RL: BIOL (Biological study)
(antinociception- and locomotion-affecting activity of, structure in relation to)

RN 90549-84-1 HCAPLUS

CN Glycine, N-[N-(N2-L-tyrosyl-D-arginyl)-L-phenylalanyl]-, ethyl ester (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L9 ANSWER 24 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1985:198185 HCAPLUS
DOCUMENT NUMBER: 102:198185
ORIGINAL REFERENCE NO.: 102:30939a,30942a
TITLE: The analgesic activity of D-Arg2-dermorphin and its N-terminal tetrapeptide analogs after subcutaneous administration in mice
AUTHOR(S): Sasaki, Y.; Matsui, M.; Fujita, H.; Hosono, M.; Taguchi, M.; Suzuki, K.; Sakurada, S.; Sato, T.; Sakurada, T.; Kisara, K.
CORPORATE SOURCE: Tohoku Coll. Pharm., Sendai, 983, Japan
SOURCE: Neuropeptides (Edinburgh, United Kingdom) (1985), 5(4-6), 391-4
CODEN: NRPPDD; ISSN: 0143-4179
DOCUMENT TYPE: Journal
LANGUAGE: English

AB 2-D-Arginine-dermorphin (I) [96425-96-6] and 19 N-terminal tetrapeptide analogs were prepared, and their analgesic activities were determined by the tail pressure test after s.c. administration in mice. The stability of a tetrapeptide I analog to enzymic degradation was also examined

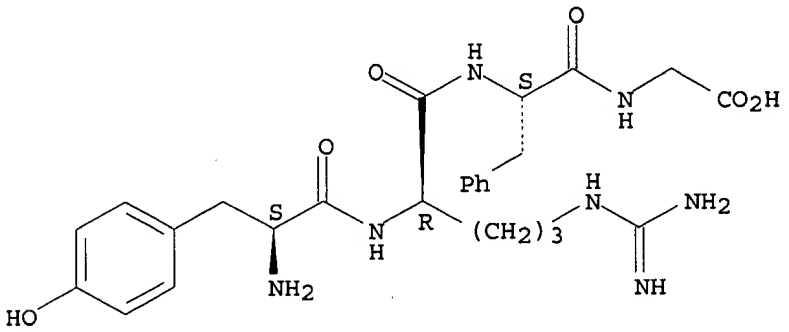
I

Updated Search

had analgesic potency equal to or slightly greater than that of dermorphin. In a series of tetrapeptide I analogs, a very pronounced activity greater than that of morphine was observed for analogs of the following structure, H-Tyr-D-Arg-Phe-X-OH (X = Gly, sarcosine, and D-Ala) and their esters. Replacement of the 2-D-arginine residue by D-nitroarginine, D-homoarginine, or D-lysine decreased the potency, suggesting that the guanidino group and side chain length of D-arginine are of great importance for a higher activity. The tetrapeptide H-Tyr-D-Arg-Phe-Gly-OH was more stable than the parent tetrapeptide (H-Tyr-D-Ala-Phe-Gly-OH) to cleavage by aminopeptidase M [9054-63-1] and carboxypeptidase Y [9046-67-7].

IT 90549-83-0
RL: BIOL (Biological study)
(analgesia from, mol. structure in relation to)
RN 90549-83-0 HCAPLUS
CN Glycine, L-tyrosyl-D-arginyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L9 ANSWER 25 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1984:584204 HCAPLUS
DOCUMENT NUMBER: 101:184204
ORIGINAL REFERENCE NO.: 101:27729a,27732a
TITLE: Comparison of the antinociceptive effect between D-Arg containing dipeptides and tetrapeptides in mice
AUTHOR(S): Sato, T.; Sakurada, S.; Sakurada, T.; Furuta, S.; Nakata, N.; Kisara, K.; Sasaki, Y.; Suzuki, K.
CORPORATE SOURCE: Dep. Pharmacol., Tohoku Coll. Pharm., Sendai, 983, Japan
SOURCE: Neuropeptides (Edinburgh, United Kingdom) (1984), 4(4), 269-79
CODEN: NRPPDD; ISSN: 0143-4179
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The D-arginine-containing dipeptides, H-Tyr-D-Arg-OMe [92758-99-1] and H-Tyr(Et)-D-Arg-OMe [92759-00-7], and D-arginine-substituted N-terminal tetrapeptides of dermorphin, H-Tyr-D-Arg-Phe-Gly-OEt [90549-84-1] and H-Tyr(Et)-D-Arg-Phe-Gly-OEt [92759-01-8] administered intracerebroventricularly exhibited dose-dependent antinociceptive activities in mice as measured by the tail-pressure and phenylbenzoquinone writhing tests. The effects of these peptides were antagonized by pretreatment with naloxone, indicating that these effects are produced through opioid receptors. The tetrapeptides were very potent (half-maximum

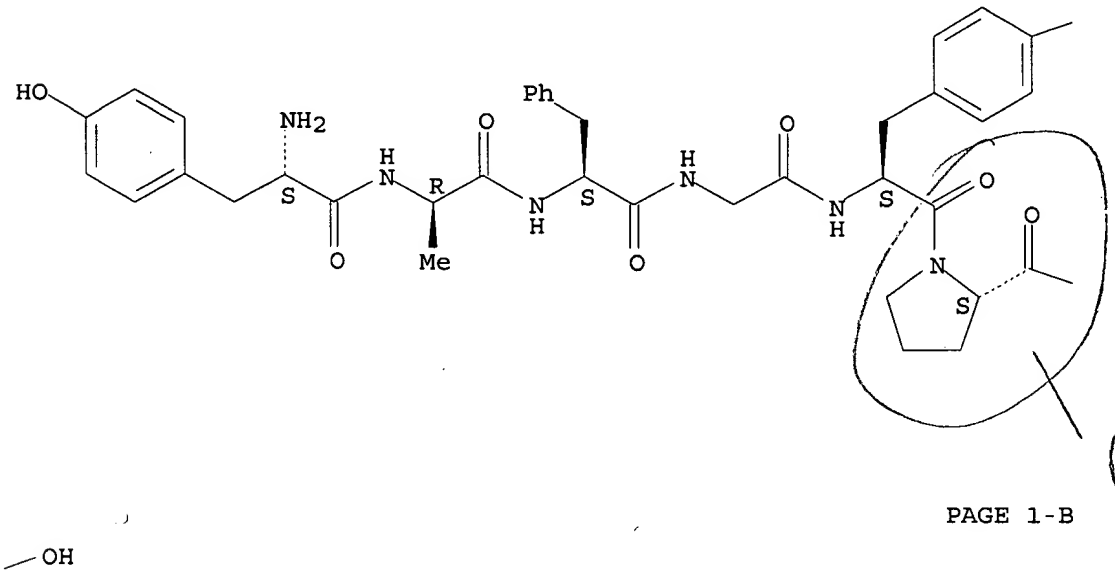
ED = 12.5 and 355.0 pmole in the tail-pressure test and 3.1 and 53.0 pmole in the phenylbenzoquinone writhing test, resp.) much more so and more prolonged than those of morphine and the dipeptides used. The difference in peak response times and the degree of antagonism by naloxone indicates that the dipeptides and tetrapeptides act on different sites in the central nervous system.

IT 77614-16-5D, analogs
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(analgesic activity of)

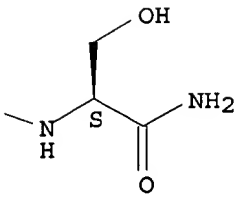
RN 77614-16-5 HCAPLUS
CN Dermorphin (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 26 OF 26 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1984:400927 HCAPLUS
DOCUMENT NUMBER: 101:927
ORIGINAL REFERENCE NO.: 101:151a,154a
TITLE: D-Arg2-dermorphin tetrapeptide analogs: a potent and long-lasting analgesic activity after subcutaneous administration

Updated Search

09890219

AUTHOR(S): Sasaki, Yusuke; Matsui, Michiko; Taguchi, Masumi; Suzuki, Kenji; Sakurada, Shinobu; Sato, Takumi ; Sakurada, Tsukasa; Kisara, Kensuke

CORPORATE SOURCE: Tohoku Coll. Pharm., Sendai, 983, Japan

SOURCE: Biochemical and Biophysical Research Communications (1984), 120(1), 214-18
CODEN: BBRCA9; ISSN: 0006-291X

DOCUMENT TYPE: Journal

LANGUAGE: English

AB To determination the pharmacol. properties of [D-Arg2]dermorphin tetrapeptides, 6 tetrapeptide analogs based on the following formulas, H-Tyr-D-Arg-Phe-Gly-OX (X = H, Et, n-propyl), H-Tyr-D-Arg-Phe-Sar-OX (X = H, Me, Et), were prepared All these analogs exhibited highly potent and long-lasting analgesia as compared with that of morphine after s.c. administration into mice. Among analogs tested, H-Tyr-D-Arg-Phe-Sar-OH showed the highest activities, which were 21, 30, and 58 times more active than morphine in the tail pressure, tail flick,and phenylbenzoquinone writhing tests, resp., on a molar basis.

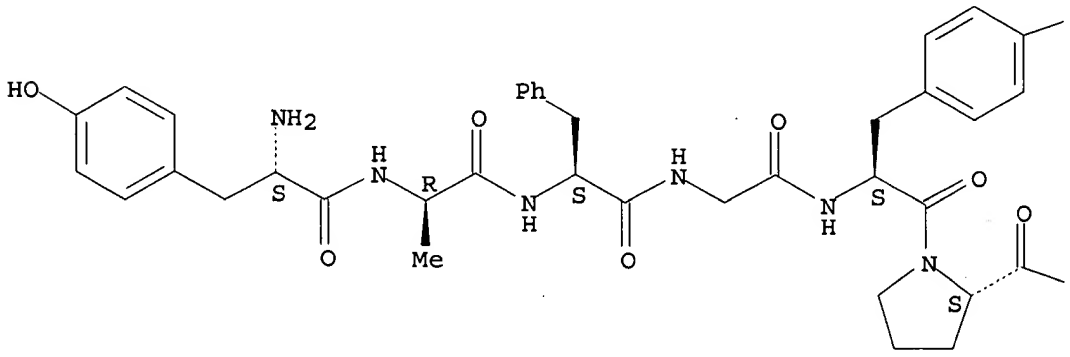
IT 77614-16-5DP, analogs
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation and analgesic activity of)

RN 77614-16-5 HCAPLUS

CN Dermorphin (CA INDEX NAME)

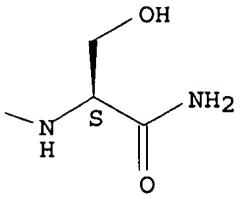
Absolute stereochemistry.

PAGE 1-A



Updated Search

OH



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L6       21413 S L5
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L12  ANSWER 1 OF 11  HCAPLUS  COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER:        2007:993589  HCAPLUS
DOCUMENT NUMBER:         147:338487
TITLE:                   Diagnostic peptides binding to a component of
                         vulnerable atherosclerotic plaques or to a solid tumor
                         component for use in non-invasive imaging
INVENTOR(S):             Kim, Young H.; Kim, Ducksoo; Rusckowski,
                         Mary
PATENT ASSIGNEE(S):      University of Massachusetts, USA
SOURCE:                  U.S. Pat. Appl. Publ., 27pp.
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Updated Search

09890219

CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2007207507	A1	20070906	US 2006-365127	20060228

PRIORITY APPLN. INFO.: US 2006-365127 20060228

AB The present invention provides compns. suitable for use as biomarkers of vulnerable plaques as well as methods for the use of such compns. In preferred embodiments, specific mol. imaging agents are provided that permit the selective identification of vulnerable plaques in coronary and other arteries using non-invasive imaging methods. Such specific mol. imaging agents comprise a binding partner linked to a detectable label that can be used in vivo to visualize vulnerable plaques. In certain preferred embodiments, the binding partner is a peptide that binds selectively to a component of a vulnerable plaque, such as myeloperoxidase or a portion thereof. In other preferred embodiments, the binding partner is an antibody that binds selectively to a component of a vulnerable plaque. In other preferred embodiments, the binding partner is a portion of a polypeptide displayed by a bacteriophage that binds selectively to a component of a vulnerable plaque. Peptide ligands for myeloperoxidase were identified in a phage display library. In another embodiment, peptide ligands bind to a solid tumor component, such as TAG-72 antigen, for non-invasive imaging of a solid tumor.

IT 908839-96-3DP, detectable label conjugates
RL: ARG (Analytical reagent use); DGN (Diagnostic use); PRP (Properties); SPN (Synthetic preparation); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence, diagnostic peptide; diagnostic peptides binding to component of vulnerable atherosclerotic plaques or to solid tumor component for use in non-invasive imaging)

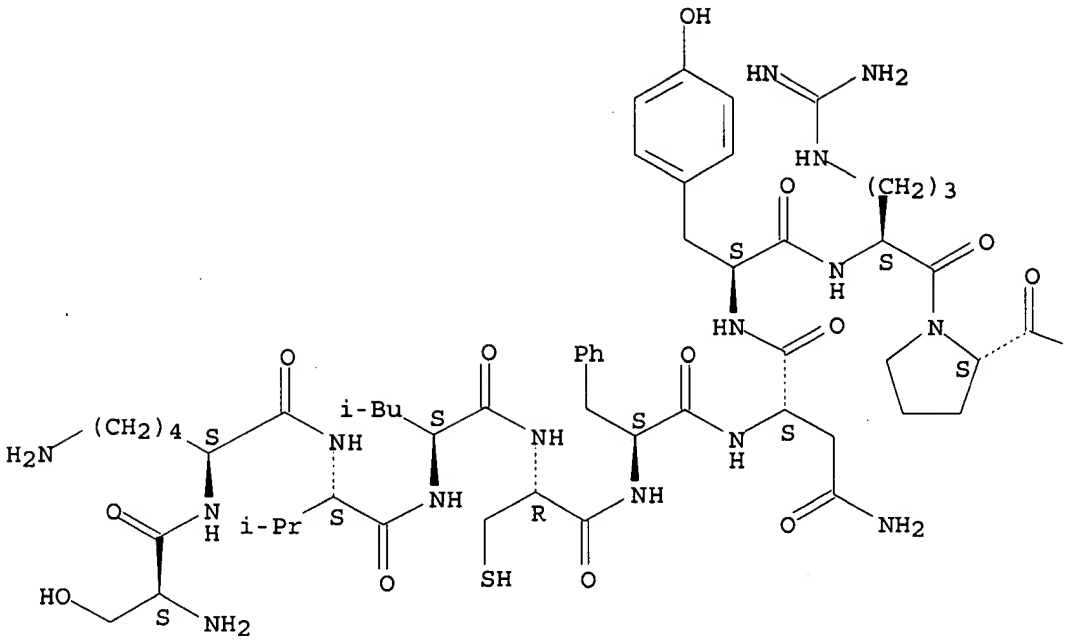
RN 908839-96-3 HCAPLUS

CN L-Cysteine, L-seryl-L-lysyl-L-valyl-L-leucyl-L-cysteinyl-L-phenylalanyl-L-asparaginyl-L-tyrosyl-L-arginyl-L-prolyl-L-threonyl- (CA INDEX NAME)

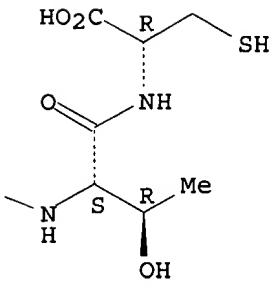
Absolute stereochemistry.

Updated Search

PAGE 1-A



PAGE 1-B



L12 ANSWER 2 OF 11 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2007:441674 HCAPLUS
DOCUMENT NUMBER: 147:73041
TITLE: Kojic acid-tripeptide amide as a new tyrosinase inhibitor
AUTHOR(S): Noh, Jin-Mi; Kwak, Seon-Yeong; Kim, Do-Hyun; Lee, Yoon-Sik
CORPORATE SOURCE: School of Chemical and Biological Engineering, Seoul National University, Seoul, 151-744, S. Korea
SOURCE: Biopolymers (2007), 88(2), 300-307

Updated Search

09890219

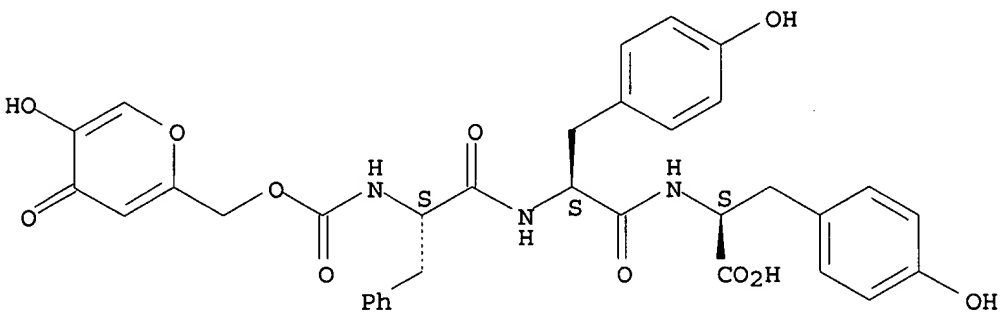
CODEN: BIPMAA; ISSN: 0006-3525
PUBLISHER: John Wiley & Sons, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Twenty two kojic acid-tripeptide amides were prepared using a solid-phase Fmoc/tBu strategy with Rink Amide SURE resin. To effectively obtain kojic acid-tripeptide amide conjugates, the coupling conditions of kojic acid to the tripeptide on the resin were optimized. The tyrosinase inhibitory activity of kojic acid-tripeptide amides and the effect of the amino acid sequence on the activity were compared with those of kojic acid-tripeptide acids. The stability of kojic acid-tripeptide amides were then compared with those of kojic acid and kojic acid-tripeptides acids. As a consequence, kojic acid-FWY-NH2 proved to be the best compound, with the highest inhibitory activity, which was maintained over different storage times under various temps. and pHs.

IT 717913-58-1
RL: COS (Cosmetic use); BIOL (Biological study); USES (Uses)
(solid-phase preparation of kojic acid-tripeptide derivs. via solid-phase peptide synthesis followed by coupling with kojic acid imidazolide, and their cosmetic property as tyrosinase inhibitors)

RN 717913-58-1 HCAPLUS
CN L-Tyrosine, N-[[(5-hydroxy-4-oxo-4H-pyran-2-yl)methoxy]carbonyl]-L-phenylalanyl-L-tyrosyl- (CA INDEX NAME)

Absolute stereochemistry.



REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 11 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2007:257615 HCAPLUS
DOCUMENT NUMBER: 146:293241
TITLE: Interactions between hnRNP H and myotonic dystrophy kinase mRNA and the development of drugs promoting kinase mRNA export in the treatment of myotonic dystrophy
INVENTOR(S): Kim, Dongho; Rossi, John J.
PATENT ASSIGNEE(S): City of Hope, USA
SOURCE: U.S. Pat. Appl. Publ., 31pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

Updated Search

US 2007054259	A1	20070308	US 2006-453260	20060615
PRIORITY APPLN. INFO.:			US 2005-691232P P	20050617

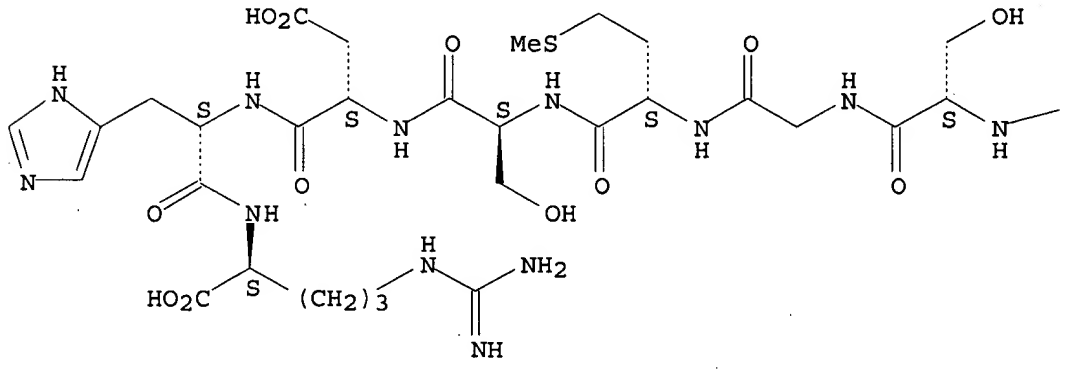
AB Heterogeneous nuclear ribonucleoprotein H (hnRNP H) is capable of binding mutant myotonic dystrophy (DM) protein kinase (DMPK) mRNA. Modulation of the expression of hnRNP H results in less nuclear retention of the mutant DMPK mRNA. Drugs modulating the export of the mRNA may therefore be useful in the treatment of type 1 myotonic dystrophy (no data.). Characterization of the interaction of hnRNP H and kinase mRNAs is described. Peptides of the hnRNP binding to the triplet repeat expansion variant mRNA are identified.

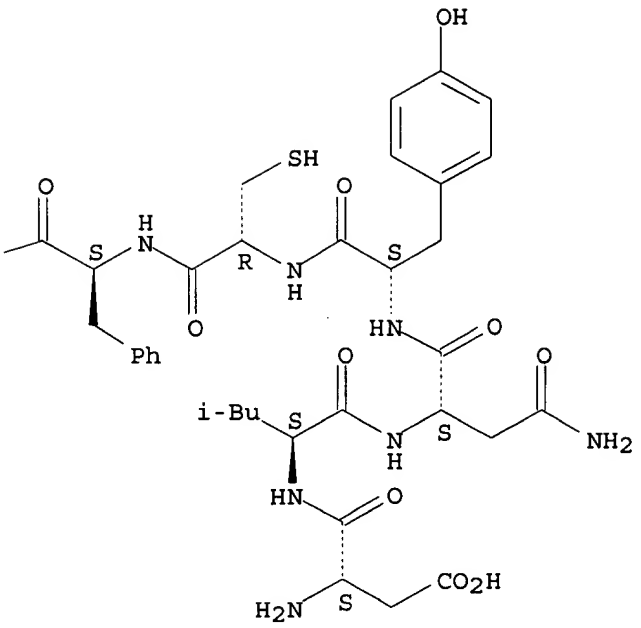
IT 927833-83-8
RL: PRP (Properties)
(unclaimed sequence; interactions between hnRNP H and myotonic dystrophy kinase mRNA and the development of drugs promoting kinase mRNA export in the treatment of myotonic dystrophy)

RN 927833-83-8 HCAPLUS

CN L-Arginine, L-α-aspartyl-L-leucyl-L-asparaginyl-L-tyrosyl-L-cysteinyl-L-phenylalanyl-L-serylglycyl-L-methionyl-L-seryl-L-α-aspartyl-L-histidyl- (CA INDEX NAME)

Absolute stereochemistry.





L12 ANSWER 4 OF 11 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2006:922133 HCAPLUS
DOCUMENT NUMBER: 145:312253
TITLE: Biomarkers of vulnerable atherosclerotic plaques and their use in detection of unstable plaques by non-invasive imaging
INVENTOR(S): Kim, Young H.; Kim, Ducksoo; Rusckowski, Nancy
PATENT ASSIGNEE(S): University of Massachusetts, USA
SOURCE: PCT Int. Appl., 49pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006093973	A2	20060908	WO 2006-US7111	20060228

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: US 2005-657111P P 20050228

Updated Search

09890219

OTHER SOURCE(S): MARPAT 145:312253

AB The present invention provides compns. suitable for use as biomarkers of unstable or vulnerable atherosclerotic plaques as well as methods for the use of such compns. In preferred embodiments, specific mol. imaging agents are provided that permit the selective identification of vulnerable plaques in coronary and other arteries using non- invasive imaging methods. Such specific mol. imaging agents comprise a binding partner linked to a detectable label that can be used in vivo to visualize vulnerable plaques. In certain preferred embodiments, the binding partner is a peptide that binds selectively to a component of a vulnerable plaque. In other preferred embodiments, the binding partner is an antibody that binds selectively to a component of a vulnerable plaque. In other preferred embodiments, the binding partner is a portion of a polypeptide displayed by a bacteriophage that binds selectively to a component of a vulnerable plaque. In preferred embodiments, the component of a vulnerable plaque is myeloperoxidase or a portion thereof. Peptide ligands for myeloperoxidase were identified in a phage display library.

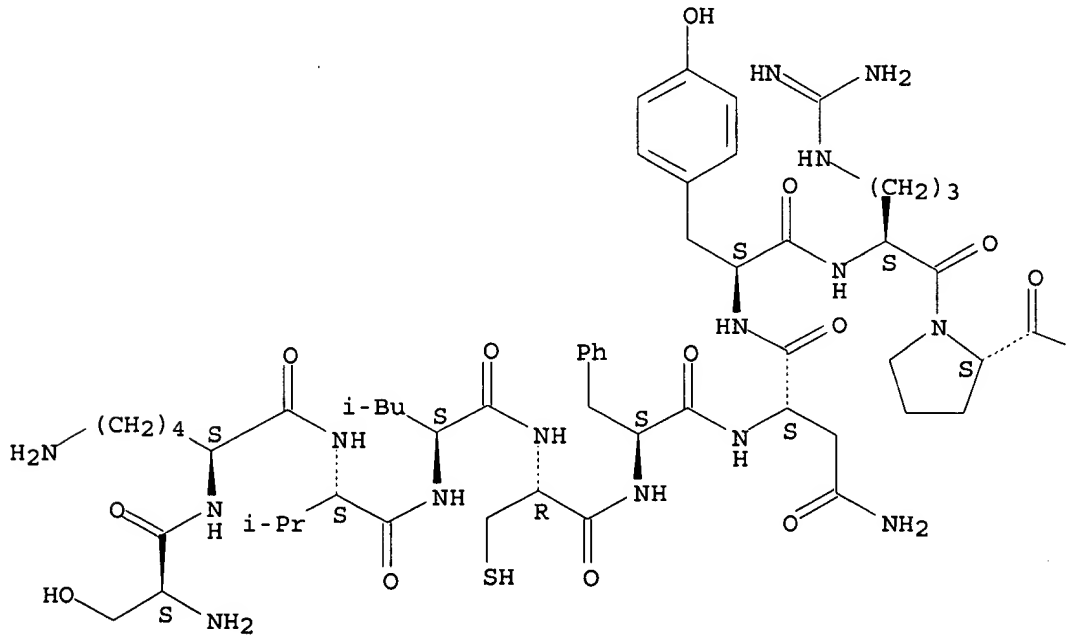
IT 908839-96-3
RL: PRP (Properties)
(unclaimed sequence; biomarkers of vulnerable atherosclerotic plaques and their use in detection of unstable plaques by non-invasive imaging)

RN 908839-96-3 HCAPLUS

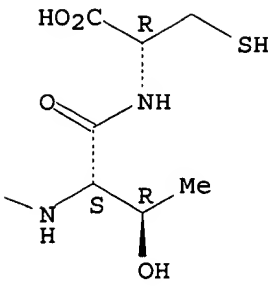
CN L-Cysteine, L-seryl-L-lysyl-L-valyl-L-leucyl-L-cysteinyl-L-phenylalanyl-L-asparaginyl-L-tyrosyl-L-arginyl-L-prolyl-L-threonyl- (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



Updated Search



L12 ANSWER 5 OF 11 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2005:216699 HCAPLUS
DOCUMENT NUMBER: 142:291343
TITLE: Methylhydrazine-derived compounds modulating β-catenin/TCF activated transcription for cancer treatment and their synthesis
INVENTOR(S): Kahn, Michael; Oh, Sewoong; Kim, Daehoon; Ha, Jongryul; Hojjati-Emami, Katayoon
PATENT ASSIGNEE(S): Choongwae Pharma Corporation, S. Korea
SOURCE: PCT Int. Appl., 174 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

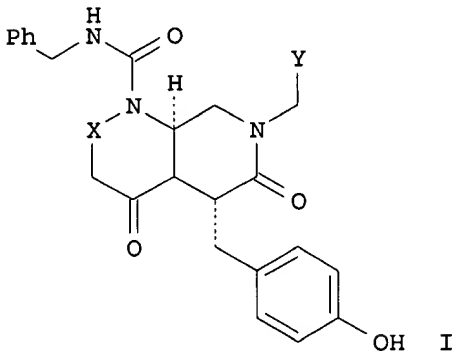
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005021025	A2	20050310	WO 2004-US28142	20040827
WO 2005021025	A3	20050707		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004268647	A1	20050310	AU 2004-268647	20040827
CA 2537099	A1	20050310	CA 2004-2537099	20040827
US 2005059628	A1	20050317	US 2004-928626	20040827
EP 1660502	A2	20060531	EP 2004-782582	20040827
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
BR 2004013969	A	20061031	BR 2004-13969	20040827
CN 1871239	A	20061129	CN 2004-80030688	20040827

Updated Search

09890219

JP 2007503816	T	20070301	JP 2006-524937		20040827
IN 2006DN00935	A	20070817	IN 2006-DN935		20060222
KR 2006121842	A	20061129	KR 2006-704085		20060227
PRIORITY APPLN. INFO.:			US 2003-498451P	P	20030828
			WO 2004-US28142	W	20040827

OTHER SOURCE(S): MARPAT 142:291343
GI



I

AB The present invention provides compds., e.g., I (X = CH2, NMe; Y = 1-naphthyl, 8-quinolinyl, 2,4-fluorophenyl) and methods for modulating transcription activated by β-catenin/TCF, such as the selective inhibition of genes targeted by the Wnt/β-catenin pathway. Specifically disclosed are four compds., i.e., Ia (I; X = CH2; Y = 1-naphthyl), Ib (I; X = NMe; Y = 1-naphthyl), Ic (I; X = NMe; Y = 8-quinolinyl), and Id (I; X = NMe; Y = 2,4-fluorophenyl), that modulate transcription activation mediated by β-catenin/TCF pathway. In particular embodiments, Compound Ia is shown to inhibit β-catenin/TCF transcription by binding to CBP (not p300) N-terminal 111-amino acid region, the minimal region in CBP for β-catenin interaction, thus disrupts the β-catenin/CBP complex (but not the p300/β-catenin complex). CBP (1-111) binds to Compound Ia in a phosphorylation independent manner. Compound Ia is demonstrated to inhibit the expression of cyclin D1, arrest cells in G1, and reduce colony growth in soft agar in a dose dependent manner. The subsets of β-catenin/TCF-responsive target genes are specifically up- or down-regulated buy Compound Ia are also provided. Compound Ic and Compound Id are shown to reduce tumor growth in min mouse model. The studies on cytotoxicity of Compound Ib and its metabolism in rat and human, and bioavailability of Compound Ib, Ic, and Id are reported.

IT 847740-18-5
RL: PRP (Properties)
(unclaimed sequence; methylhydrazine-derived compds. modulating β-catenin/TCF activated transcription for cancer treatment and their synthesis)

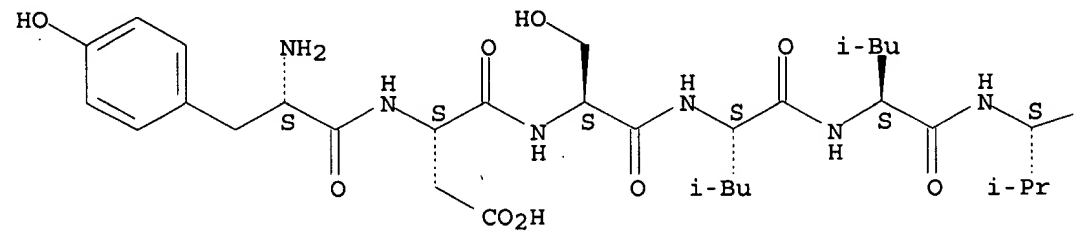
RN 847740-18-5 HCAPLUS

CN L-Leucine, L-tyrosyl-L-α-aspartyl-L-seryl-L-leucyl-L-leucyl-L-valyl-L-phenylalanyl-L-α-aspartyl-L-tyrosyl-L-α-glutamylglycyl-L-serylglycyl-L-seryl-L-alanyl-L-alanyl-L-seryl-L-leucyl-L-seryl-L-seryl- (9CI) (CA INDEX NAME)

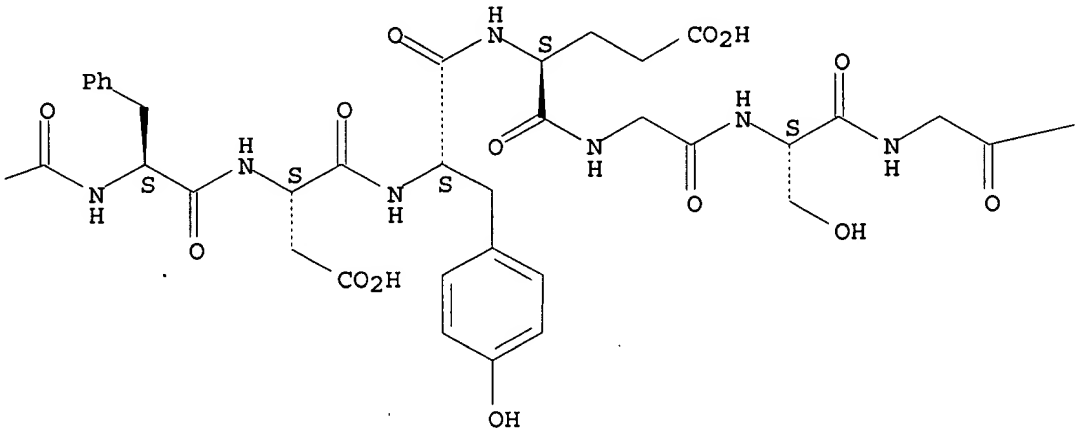
Absolute stereochemistry.

Updated Search

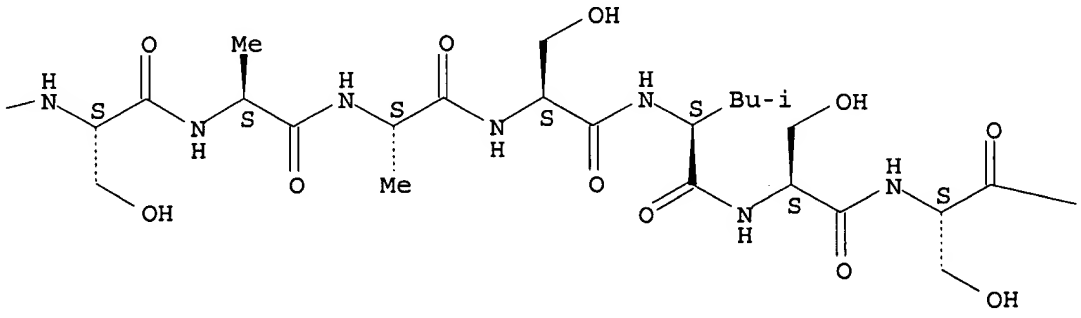
PAGE 1-A



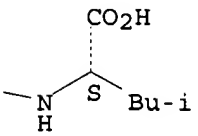
PAGE 1-B



PAGE 1-C



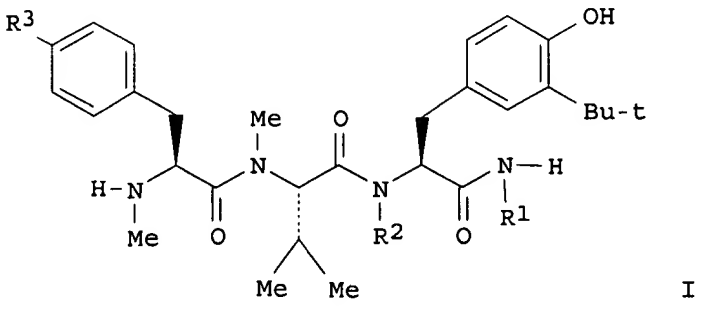
Updated Search



L12 ANSWER 6 OF 11 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2002:637704 HCAPLUS
DOCUMENT NUMBER: 137:185838
TITLE: Process for preparation of peptide derivatives
INVENTOR(S): Kim, Dong Ick; Jeon, Gee Ho; Kim, Sung Jin
PATENT ASSIGNEE(S): Chugai Seiyaku Kabushiki Kaisha, Japan
SOURCE: PCT Int. Appl., 40 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002064623	A1	20020822	WO 2002-JP1139	20020212
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002230216	A1	20020828	AU 2002-230216	20020212
PRIORITY APPLN. INFO.:			KR 2001-6673 A	20010212
			WO 2002-JP1139 W	20020212

OTHER SOURCE(S): CASREACT 137:185838; MARPAT 137:185838
GI



I

Updated Search

09890219

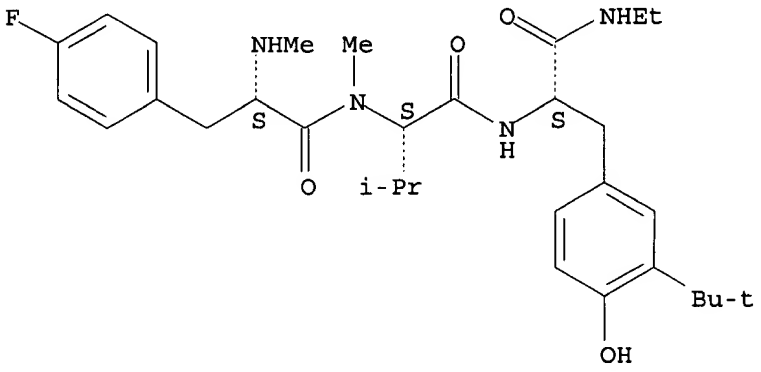
AB The title compds. I [R1 is hydrogen or linear or branched C1-4 alkyl; R2 is hydrogen or linear or branched C1-4 alkyl; and R3 is halogeno] are prepared in a multistep process. I are motilin receptor antagonists and are useful as drugs for gastric or intestinal diseases (no data). Thus, amidation of N-(tert-butoxycarbonyl)-L-(4-fluorophenyl)alanine with L-valine Me ester hydrochloride, followed by methylation with iodomethane, saponification, reaction with 3-tert-butyl-L-tyrosine Et amide, and deprotection, gave N-methyl-L-4-fluorophenylalanyl-N-methyl-L-valine-3-tert-butyl-L-tyrosine Et amide.

IT 287206-61-5P
RL: IMF (Industrial manufacture); PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(process for preparation of peptide derivs.)

RN 287206-61-5 HCAPLUS

CN L-Tyrosinamide, 4-fluoro-N-methyl-L-phenylalanyl-N-methyl-L-valyl-3-(1,1-dimethylethyl)-N-ethyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 7 OF 11 HCAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2001:348247 HCAPLUS

DOCUMENT NUMBER: 135:88982

TITLE: Purification and characterization of acharan sulfate lyases, two novel heparinases, from Bacteroides stercoris HJ-15

AUTHOR(S): Kim, Byung-Taek; Hong, Sung-Woon; Kim, Wan-Seok; Kim, Yeong Shik; Kim, Dong-Hyun

CORPORATE SOURCE: College of Pharmacy, Kyung Hee University, Seoul, 130-701, S. Korea

SOURCE: European Journal of Biochemistry (2001), 268(9), 2635-2641
CODEN: EJBCAI; ISSN: 0014-2956

PUBLISHER: Blackwell Science Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Two novel acharan sulfate lyases (ASL1 and ASL2: no EC number) have been purified from Bacteroides stercoris HJ-15 which was isolated from human intestinal bacteria with glycosaminoglycan (GAG) degrading enzymes. These

Updated Search

enzymes were purified to apparent homogeneity by a combination of QAE-cellulose, DEAE-cellulose, carboxymethyl-Sephadex C-50, hydroxyapatite and HiTrap SP Sephadex C-25 column chromatog. with the final specific activity of 50.5 and 76.7 μmol·min-1·mg-1, resp. Both acharan sulfate lyases are single subunits of 83 kDa by SDS-PAGE and gel filtration. ASL1 showed optimal activity at pH 7.2 and 45°. ASL1 activity was inhibited by Cu2+, Ni2+ and Co2+, but ASL2 activity was inhibited by Cu2+, Ni2+ and Pb2. Both enzymes were slightly inhibited by some agents that modify histidine and cysteine residues, but activated by reducing agents such as DL-dithiothreitol and 2-mercaptoethanol. Both purified bacteroidal acharan sulfate lyases acted to the greatest extent on acharan sulfate, and to a lesser extents on heparan sulfate and heparin. They did not act on de-O-sulfated acharan sulfate. These findings suggest that the biochem. properties of these purified acharan sulfate lyases are different from those of the previously purified heparin lyases, but these enzymes belong to heparinase II.

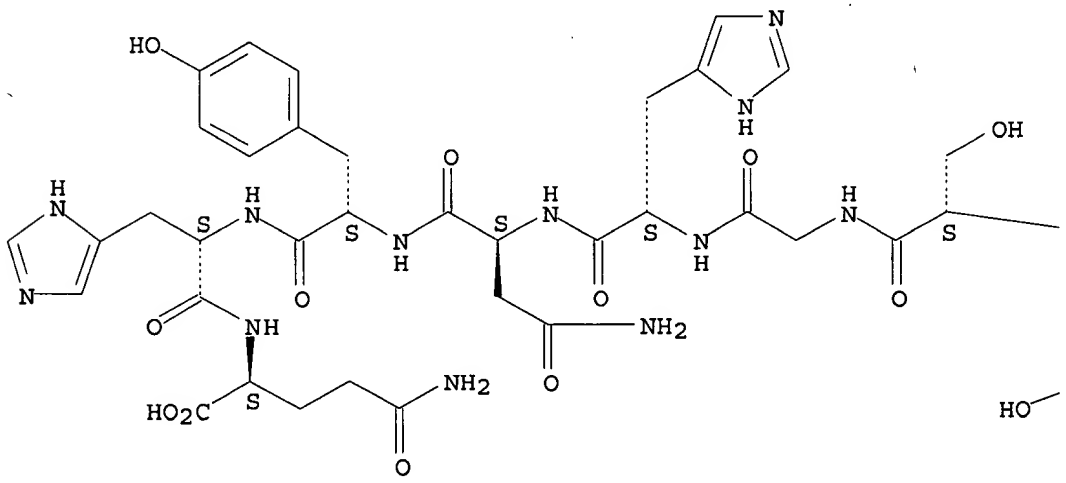
IT 349487-50-9
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(internal amino-acid sequence of acharan sulfate lyase 1, a novel heparinase, from Bacteroides stercoris HJ-15)

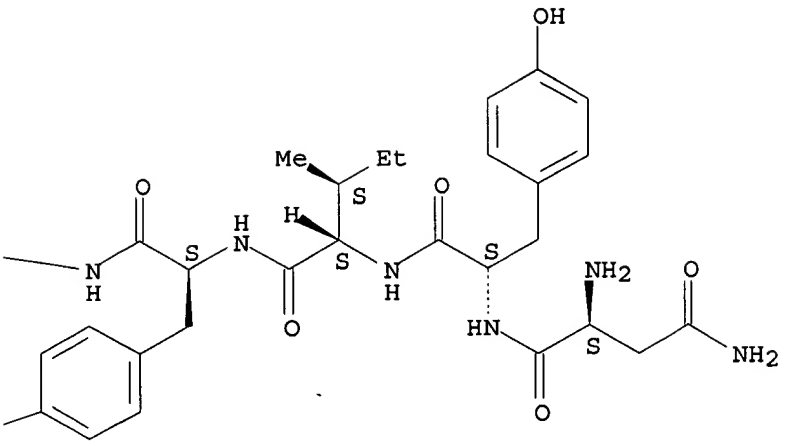
RN 349487-50-9 HCAPLUS

CN L-Glutamine, L-asparaginyl-L-tyrosyl-L-isoleucyl-L-tyrosyl-L-serylglycyl-L-histidyl-L-asparaginyl-L-tyrosyl-L-histidyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



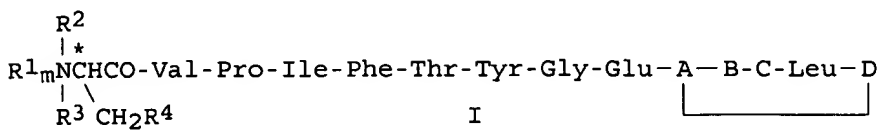


REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 8 OF 11 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1997:761836 HCAPLUS
DOCUMENT NUMBER: 127:359107
TITLE: Preparation of motilin-like cyclopeptides with gastrointestinal motor stimulating activity
INVENTOR(S): Dharanipragada, Ramalinga; Macielag, Mark J.; Kim-Dettelback, Jung; Florance, James
PATENT ASSIGNEE(S): Ohmeda Pharmaceutical Products Division Inc., USA
SOURCE: Eur. Pat. Appl., 25 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 807639	A2	19971119	EP 1997-303251	19970513
EP 807639	A3	19971126		
EP 807639	B1	20000209		
R: AT, BE, CH, DE, DK, ES, FR, GB, IT, LI, LU, NL, SE, IE				
US 5734012	A	19980331	US 1996-648644	19960516
CA 2200935	A1	19971116	CA 1997-2200935	19970325
CA 2200935	C	20050524		
AT 189685	T	20000215	AT 1997-303251	19970513
ES 2144826	T3	20000616	ES 1997-303251	19970513
JP 10053599	A	19980224	JP 1997-126736	19970516
PRIORITY APPLN. INFO.:			US 1996-648644 A	19960516

OTHER SOURCE(S): MARPAT 127:359107
GI



I

Updated Search

09890219

AB This invention pertains to cyclic polypeptides I [R1 = alkyl; R2 = H, alkyl; R3 = H, alkyl; R4 = Ph, Ph substituted with 1 or more halo, OH, or alkoxy substituents; R5 = OH, NH2; A = L-Glu, L-Asp, L-Lys, L-Orn, L-2,4-diaminobutyric acid; B = L-Ala, L-Gln; C = L-Arg, D-Arg; D = L-Lys, L-Orn, L-2,4-diaminobutyric acid, L-Glu, L-Asp; E = bond, L-Lys, D-Lys; m = 0, 1; * = asym. C which may be in the D- or L-configuration; with provisos that (a) when A = L-Glu or L-Asp, D = L-Lys, L-Orn, L-2,4-diaminobutyric acid; and (b) when A = L-Lys, L-Orn, L-2,4-diaminobutyric acid, D = L-Glu or L-Asp] including optically active isomeric forms and pharmaceutically acceptable acid addition salts thereof, having gastrointestinal motor stimulating activity. This invention also pertains to methods for using the novel cyclic polypeptides. Thus, cyclo10,14[Asp10,Leu13,Lys14]motilin(1-14) amide (porcine) (I; m = 0, R2 = R3 = H, R4 = Ph, A = Asp, B = Gln, C = Arg, D = Lys, E = bond, R5 = NH2; * = L-configuration) (II) was prepared as its bis(trifluoroacetate) salt by standard solid-phase methods on an MBHA resin using N-tert-butoxycarbonyl (Boc) amino acids. II and related cyclopeptides were tested as motilin receptor agonists in binding and contractility expts.

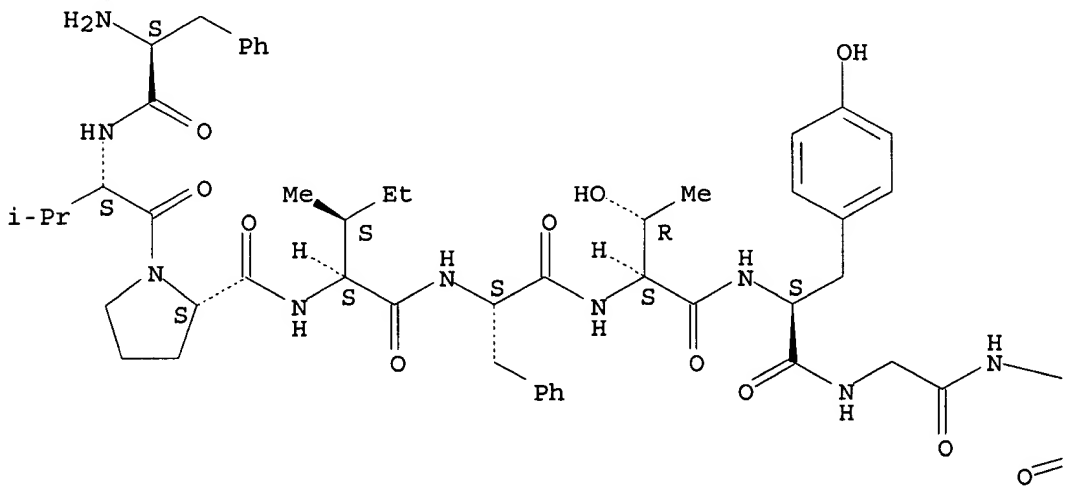
IT 59530-69-7P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of motilin-like cyclopeptides with gastrointestinal motor stimulating activity)

RN 59530-69-7 HCAPLUS

CN Motilin (swine), 13-L-leucine- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



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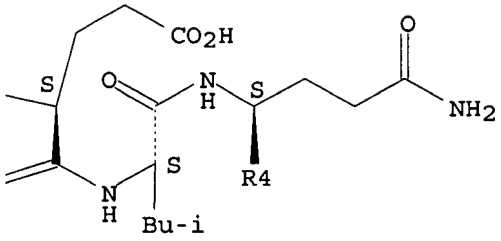

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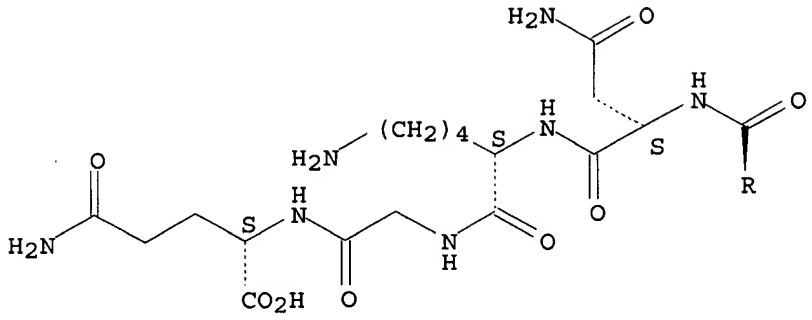

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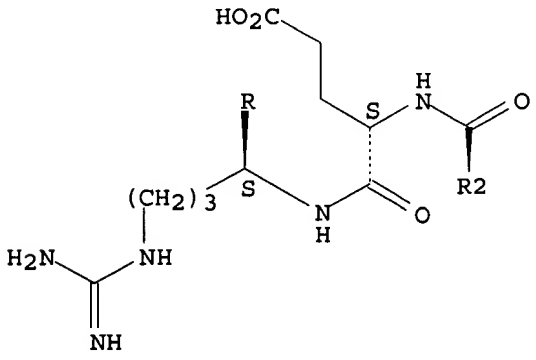

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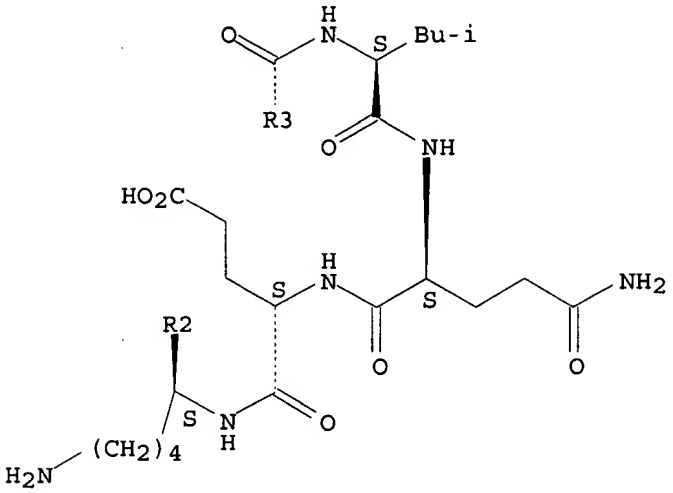

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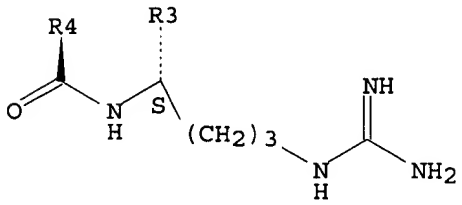


L12 ANSWER 9 OF 11 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1996:696044 HCAPLUS
DOCUMENT NUMBER: 126:26942
TITLE: Substitution of Pro3 in [Leu13]motilin affords antagonists to the GI motilin receptor
AUTHOR(S): Macielag, M. J.; Depoortere, I.; Florance, J. R.; Peeters, T. L.; Dharanipragada, R.; Kim-Dettelback, J.; Marvin, M. S.; Galdes, A.
CORPORATE SOURCE: Ohmeda PPD, New Providence, NJ, 07974, USA
SOURCE: Peptides: Chemistry, Structure and Biology, Proceedings of the American Peptide Symposium, 14th, Columbus, Ohio, June 18-23, 1995 (1996), Meeting Date 1995, 659-660. Editor(s): Kaumaya, Pravin T. P.; Hodges, Robert S. Mayflower Scientific: Kingswinford, UK.
CODEN: 63NTAF
DOCUMENT TYPE: Conference
LANGUAGE: English
AB The proline residue at position 3 of [Leu13]motilin(1-14) was systematically modified in order to elucidate the physicochem. and conformational factors leading to motilin agonism and antagonism.
IT 138143-07-4
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)

Updated Search

09890219

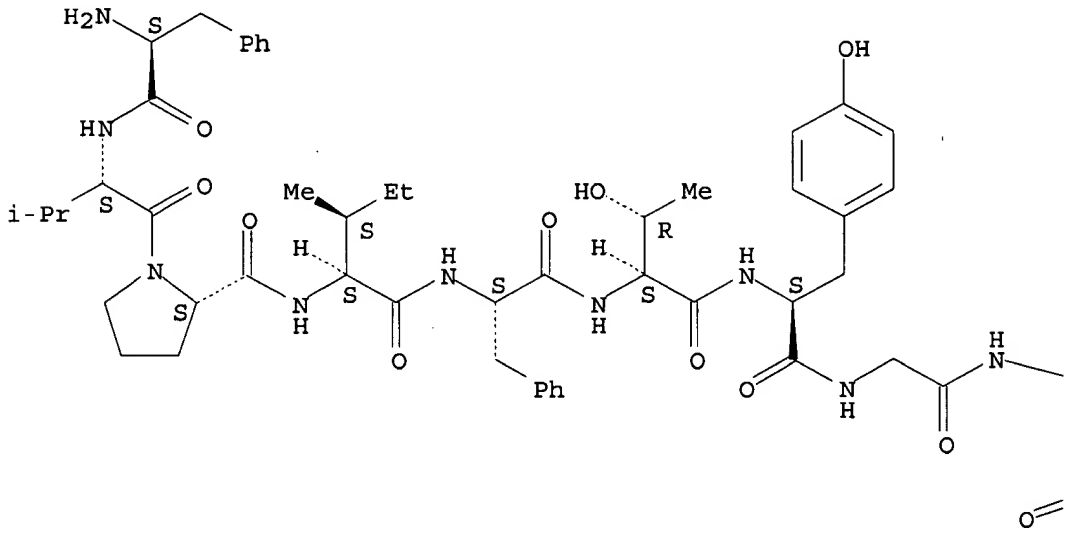
(substitution of Pro3 in [Leu13]motilin affords antagonists to GI motilin receptor)

RN 138143-07-4 HCAPLUS

CN 1-14-Motilin (swine), 13-L-leucine- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



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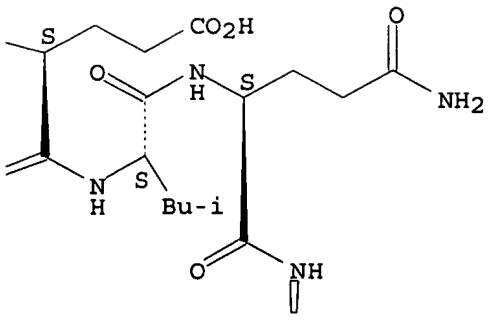

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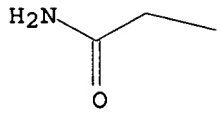

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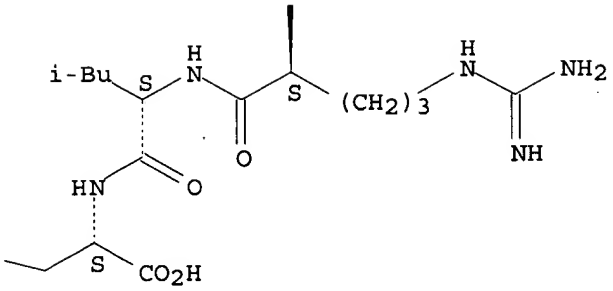


L12 ANSWER 10 OF 11 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1994:124023 HCAPLUS
DOCUMENT NUMBER: 120:124023
TITLE: A correlation between the permeability characteristics of a series of peptides using an in vitro cell culture model (Caco-2) and those using an in situ perfused rat ileum model of the intestinal mucosa
AUTHOR(S): Kim, Dong Chool; Burton, Philip S.; Borchardt, Ronald T.
CORPORATE SOURCE: Dep. Pharm. Chem., Univ. Kansas, Lawrence, KS, 66045, USA
SOURCE: Pharmaceutical Research (1993), 10(12), 1710-14
CODEN: PHREEB; ISSN: 0724-8741
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In an attempt to establish an in vitro/in situ correlation of intestinal permeability data, the permeability coeffs. (Papp) for a series of model peptides, which were determined using an in situ perfused rat ileum model, were compared to the permeability coeffs. (Pmono) determined using an in vitro cell culture model (Caco-2). The model peptides, which were all blocked on the N-terminal (acetyl, Ac) and the C-terminal (amide, NH2) ends, consisted of D-phenylalanine (F) residues (e.g., AcFNH2, AcFFNH2, AcFFFNH2). To alter the degree of hydrogen bonding potential, the nitrogens of the amide bonds were sequentially methylated [e.g., AcFF(Me)FNH2, AcF(Me)F(Me)FNH2, Ac(Me)F(Me)F(Me)FNH2, Ac(Me)F(Me)F(Me)FNH(Me)]. These peptides were shown not to be metabolized in the in situ perfused rat ileum system. The results of the transport expts. showed that there were poor correlations

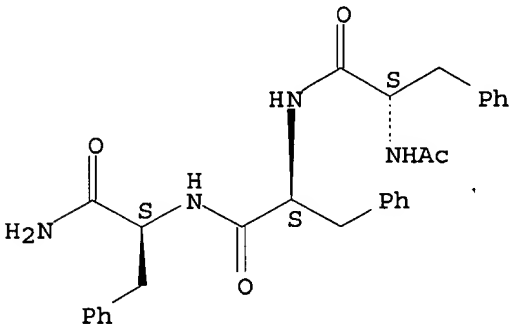
between the apparent permeability coeffs. (Papp) determined in an in situ perfused rat ileum model and the octanol-water partition coeffs. (r = 0.60) or the hydrogen bonding nos. (r = 0.63) of these peptides. However, good correlations were observed between the in situ Papp values for these peptides and their partition coeffs. in heptane-ethylene glycol (r = 0.96) and the differences in their partition coeffs. between octanol-water and iso-octane-water (r = 0.86). These results suggest that lipophilicity may not be the major factor in determining the intestinal permeability of these peptides and that hydrogen bonding potential may be a major contributing factor. A good correlation (r = 0.94) was also observed between the Papp values determined for these peptides in the in situ perfused ileum model and those Pmono values determined in the in vitro cell culture model (Caco-2) (Conradi et al., 1991). These results suggest that the permeability values determined in the Caco-2 cell culture model may be a good predictor of the intestinal permeability of peptides.

IT 152971-76-1
RL: PRP (Properties)
(intestinal permeability of, Caco-2 cells as model for)

RN 152971-76-1 HCAPLUS

CN L-Phenylalaninamide, N-acetyl-L-phenylalanyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L12 ANSWER 11 OF 11 HCAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1994:101669 HCAPLUS

DOCUMENT NUMBER: 120:101669

TITLE: Hypovirulence-associated traits induced by a mycovirus of Cryphonectria parasitica are mimicked by targeted inactivation of a host gene

AUTHOR(S): Zhang, Lei; Churchill, Alice C. L.; Kazmierczak, Pam; Kim, Dae Hyuk; van Alfen, Neal K.

CORPORATE SOURCE: Dep. Plant Pathol. Microbiol., Texas A and M Univ., College Station, TX, 77843, USA

SOURCE: Molecular and Cellular Biology (1993), 13(12), 7782-92
CODEN: MCEBD4; ISSN: 0270-7306

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Expression of the Vir2 gene of Cryphonectria parasitica is down-regulated in strains of the fungus containing a double-stranded RNA genetic element that reduces fungal virulence (W. A. Powell and N. K. Van Alfen, 1987). The Vir2 gene was now sequenced and its structure characterized; the mRNA contains a short open reading frame whose product has structural similarities to several fungal pheromones. A null mutant was constructed

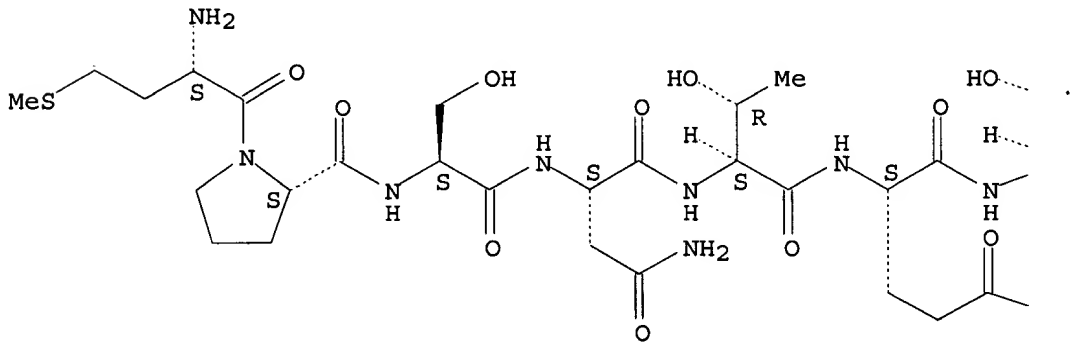
by homologous recombination to determination the function of the Vir2 gene and whether its disruption resulted in any of the altered phenotypes exhibited by many hypovirulent strains, such as redns. in virulence, pigmentation, and sporulation. The Vir2 null mutant (18dm) exhibited a wild-type phenotype with respect to gross colony morphol., growth rate, pigmentation, asexual spore viability, and virulence in apple fruit and chestnut trees. However, nos. of asexual fruiting bodies (pycnidia) and conidia were reduced significantly in comparison with the wild-type strain EP155/2. In sexual crosses of 18dm with a wild-type strain of the opposite mating type, perithecia (sexual fruiting bodies) developed but were barren. Deletion of the Vir2 gene results in a phenotype that mimics that of many double-stranded-RNA-containing hypovirulent strains; i.e., the null mutant exhibits significant redns. in asexual sporulation and pycinidium production as well as impaired sexual crossing ability. This is the first report of the partial reproduction of a virus-induced phenotype by deletion of a virus-perturbed host gene.

IT 152873-54-6, Peptide (Cryphonectria parasitica strain 155/2 clone pWPB gene vir2)
RL: PRP (Properties)
(amino acid sequence and function of, hypovirulence -associated traits of myxovirus in relation to)

RN 152873-54-6 HCAPLUS

CN L-Methionine, L-methionyl-L-prolyl-L-seryl-L-asparaginyl-L-threonyl-L-glutaminyl-L-threonyl-L-seryl-L-asparaginyl-L-seryl-L-seryl-L-methionylglycyl-L-valyl-L-asparaginylglycyl-L-tyrosyl-L-seryl-L-tyrosyl-L-cysteinyl-L-valyl-L-valyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



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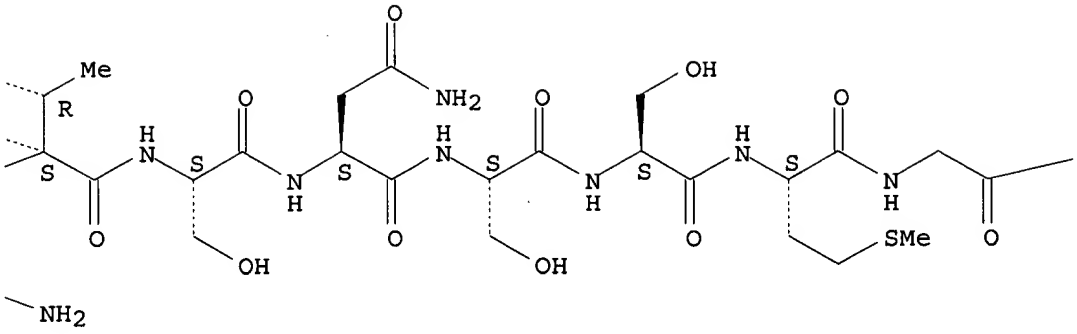

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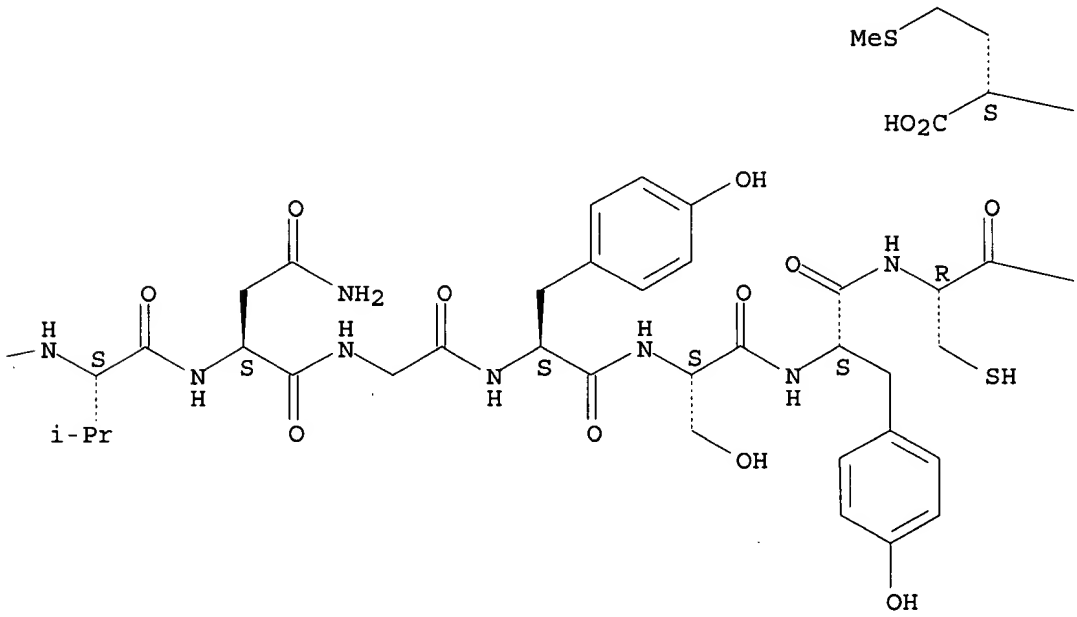
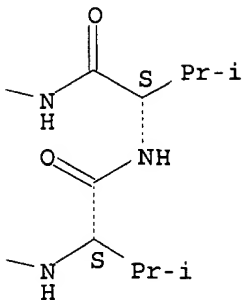


Updated Search



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L5        58230 S L3 FULL

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L12          11 S L10 AND KIM, D?/AU

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L15  ANSWER 1 OF 5  HCAPLUS  COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER:         2007:1312028  HCAPLUS
DOCUMENT NUMBER:          148:9354
TITLE:                    Regulation of functional phenotypes of cord
                          blood-derived eosinophils by γ-secretase
                          inhibitor
AUTHOR(S):                Kang, Jin Hyun; Lee, Da Hye; Seo, Hyemyung; Park, Jong
                          Sook; Nam, Key Hyun; Shin, Soon Young; Park,
                          Choon-Sik; Chung, Il Yup
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Updated Search

09890219

CORPORATE SOURCE: Division of Molecular and Life Sciences, College of Science and Technology, Hanyang University, Ansan, S. Korea

SOURCE: American Journal of Respiratory Cell and Molecular Biology (2007), 37(5), 571-577
CODEN: AJRBEL; ISSN: 1044-1549
PUBLISHER: American Thoracic Society
DOCUMENT TYPE: Journal
LANGUAGE: English

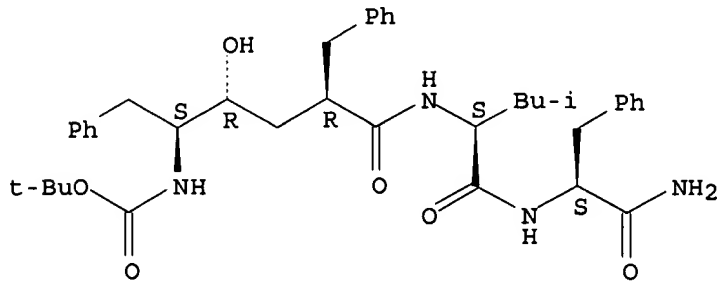
AB Eosinophils develop from stem cells in the bone marrow under the influence of hematopoietic cytokines, particularly IL-5. Previously, we have demonstrated that blockage of Notch signaling by a γ-secretase inhibitor (GSI) promotes the differentiation of umbilical cord blood (UCB)-derived eosinophils. These highly major basic protein (MBP)-pos. eosinophils cultured in the presence of the inhibitor lack the migratory response to eotaxin, although their CCR3 levels are similar to those of eosinophils cultured without the inhibitor. We investigated the mechanism underlying the differential responses of differentiating eosinophils and their functionalities in response to eosinophil-active cytokines in the presence and absence of GSI. UCB cells cultured for 4 wk with hematopoietic cytokines in the presence or absence of GSI were monitored for extracellular signal-regulated kinase (ERK) phosphorylation, MBP expression, and functionality. Eosinophil differentiation from UCB cells was accompanied by activation of the ERK1/2 pathway during the 4-wk culture period. In particular, strong ERK1/2 phosphorylation was observed in eosinophils during the final stage of culture when GSI was present. Consistent with this finding, ERK inhibition nullified the effect of GSI on eosinophil differentiation. Eosinophils cultured with GSI resembled airway eosinophils rather than peripheral blood eosinophils based on reduced IL-5Rα expression, blunted eosinophil cationic protein (ECP) degranulation, and decreased IL-13 and granulocyte macrophage-colony-stimulating factor production These results suggest that Notch signaling regulates the terminal differentiation and subsequent effector phenotypes of eosinophils, partly through modulation of the ERK pathway. GSI has therapeutic potential for eosinophilic inflammatory diseases, such as asthma.

IT 292632-98-5, L685458
RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); BIOL (Biological study)
(regulation of functional phenotypes of cord blood-derived eosinophils by γ-secretase inhibitor)

RN 292632-98-5 HCAPLUS

CN L-Phenylalaninamide, N-[(2R,4R,5S)-5-[[(1,1-dimethylethoxy)carbonyl]amino]-4-hydroxy-1-oxo-6-phenyl-2-(phenylmethyl)hexyl]-L-leucyl- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



Updated Search

09890219

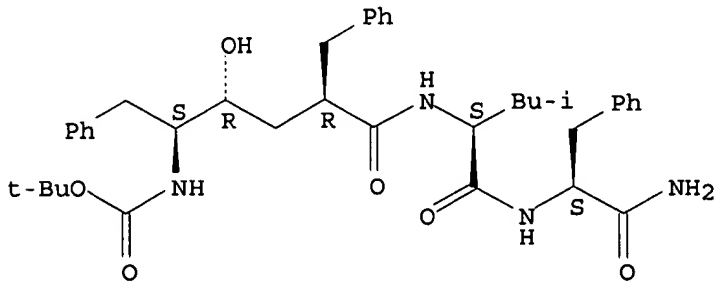
L15 ANSWER 2 OF 5 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2005:1160563 HCAPLUS
DOCUMENT NUMBER: 143:458427
TITLE: Eosinophilic differentiation is promoted by blockage of Notch signaling with a gamma-secretase inhibitor
AUTHOR(S): Kang, Jin Hyun; Lee, Da Hye; Lee, Jong Soo; Kim, Hai Joong; Shin, Jin Woo; Lee, Young Han; Lee, Young Seek; Park, Choon-Sik; Chung, Il Yup
CORPORATE SOURCE: Division of Molecular and Life Sciences, College of Science and Technology, Hanyang University, Ansan, S. Korea
SOURCE: European Journal of Immunology (2005), 35(10), 2982-2990
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: Wiley-VCH Verlag GmbH & Co. KGaA
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Although increasing evidence supports the inhibitory role of Notch in granulocyte differentiation, the direct effects of Notch on the differentiation and maturation of eosinophils, one type of granulocyte, have not yet been studied. We investigated whether a blockage of Notch signaling promoted the differentiation of eosinophils from umbilical cord blood (UCB) cells. Freshly isolated UCB cells were cultured with IL-3, IL-5 and GM-CSF in the presence or absence of a γ-secretase inhibitor L-685, 458, and examined for the expression of major basic protein (MBP). Freshly isolated UCB cells expressed mRNA and proteins for Notch 1, Notch 2, Delta 1, and Jagged 1. MBP expression in cultures with the inhibitor was significantly increased, as compared with the cultures in the absence of the inhibitor. Treatment with the inhibitor was accompanied by a decrease in Hes 1 mRNA expression, indicative of Notch-mediated signaling for the inhibitor effect. UCB cells cultured with the inhibitor for 28 days displayed similar levels of CCR3, a late marker of eosinophil development, as compared with the cells cultured without the inhibitor, but almost completely lost chemotaxis response to eotaxin. Our data suggest that Notch signaling may modulate eosinophil migration at the mature stage as well as inhibit eosinophil differentiation.

IT 292632-98-5, L 685458
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(Notch signaling blocked by γ-secretase inhibitor L-685,458)

RN 292632-98-5 HCAPLUS
CN L-Phenylalaninamide, N-[(2R,4R,5S)-5-[[(1,1-dimethylethoxy)carbonyl]amino]-4-hydroxy-1-oxo-6-phenyl-2-(phenylmethyl)hexyl]-L-leucyl- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



Updated Search

09890219

REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 3 OF 5 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2002:478996 HCAPLUS
DOCUMENT NUMBER: 137:197250
TITLE: Identification of calmodulin isoform-specific binding peptides from a phage-displayed random 22-mer peptide library
AUTHOR(S): Choi, Ji Young; Lee, Sang Hyoung; Park, Chan Young; Do Heo, Won; Kim, Jong Cheol; Kim, Min Chul; Chung, Woo Sik; Moon, Byeong Cheol; Cheong, Yong Hwa; Kim, Cha Young; Yoo, Jae Hyuk; Koo, Ja Choon; Ok, Hyun Mi; Chi, Seung-Wook; Ryu, Seong-Eon; Lee, Sang Yeol; Lim, Chae Oh; Cho, Moo Je
CORPORATE SOURCE: Plant Molecular Biology and Biotechnology Research Center, Gyeongsang National University, Jinju, 660-701, S. Korea
SOURCE: Journal of Biological Chemistry (2002), 277(24), 21630-21638
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Plants express numerous calmodulin (CaM) isoforms that exhibit differential activation or inhibition of CaM-dependent enzymes in vitro; however, their specificities toward target enzyme/protein binding are uncertain. A random peptide library displaying a 22-mer peptide on a bacteriophage surface was constructed to screen peptides that specifically bind to plant CaM isoforms (soybean calmodulin (ScaM)-1 and SCaM-4 were used in this study) in a Ca2+-dependent manner. The deduced amino acid sequence analyses of the resp. 80 phage clones that were independently isolated via affinity panning revealed that SCaM isoforms require distinct amino acid sequences for optimal binding. SCaM-1-binding peptides conform to a 1-5-10 ((FILVW)XXX(FILV)XXXX(FILVW)) motif (where X denotes any amino acid), whereas SCaM-4-binding peptide sequences conform to a 1-8-14 ((FILVW)XXXXXX(FAILVW)XXXXX(FILVW)) motif. These motifs are classified based on the positions of conserved hydrophobic residues. To examine their binding properties further, two representative peptides from each of the SCaM isoform-binding sequences were synthesized and analyzed via gel mobility shift assays, Trp fluorescent spectra analyses, and phosphodiesterase competitive inhibition expts. The results of these studies suggest that SCaM isoforms possess different binding sequences for optimal target interaction, which therefore may provide a mol. basis for CaM isoform-specific function in plants. Furthermore, the isolated peptide sequences may serve not only as useful CaM-binding sequence refs. but also as potential reagents for studying CaM isoform-specific function in vivo.

IT 454487-16-2
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(identification of calmodulin isoform-specific binding peptides from a phage-displayed random 22-mer peptide library)

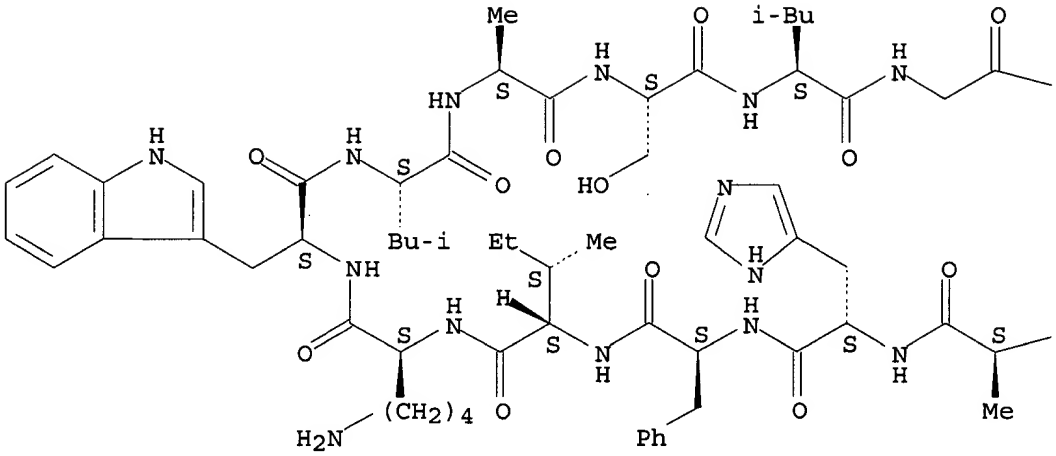
RN 454487-16-2 HCAPLUS
CN L-Leucine, L-alanyl-L-prolyl-L-alanyl-L-histidyl-L-phenylalanyl-L-isoleucyl-L-lysyl-L-tryptophyl-L-leucyl-L-alanyl-L-seryl-L-leucylglycyl-L-phenylalanyl-L-valyl-L-tyrosyl-L-leucyl-L-prolyl-L-arginyl-L-threonyl-L-seryl- (9CI) (CA INDEX NAME)

Updated Search

09890219

Absolute stereochemistry.

PAGE 1-A



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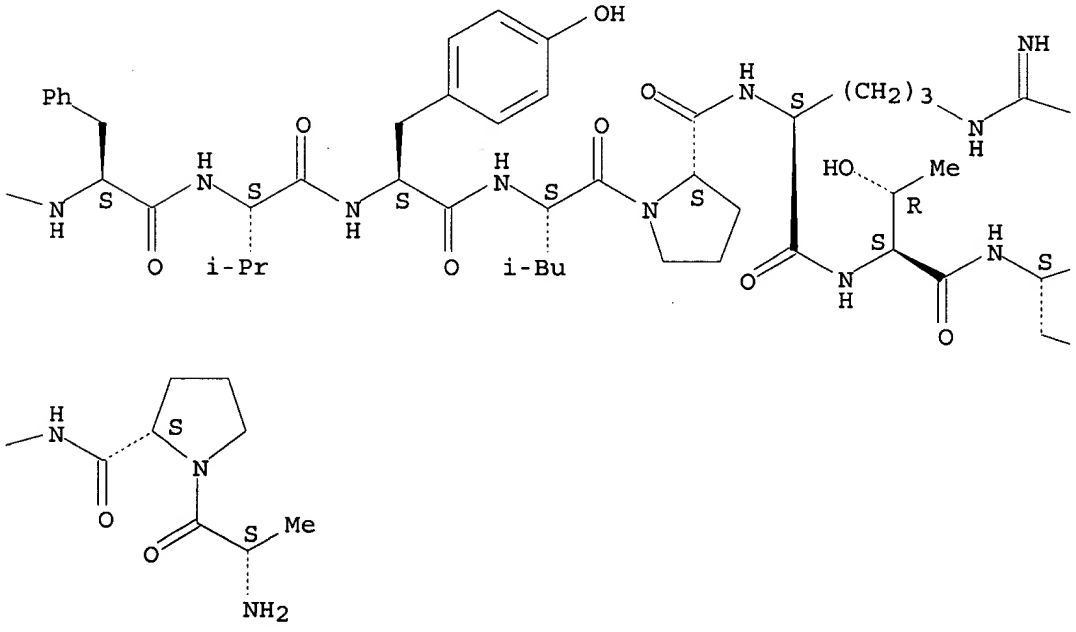
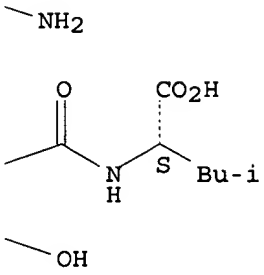


Updated Search



REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 4 OF 5 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2001:98463 HCAPLUS
DOCUMENT NUMBER: 134:161872
TITLE: Crude extract from Viscum album coloratum, and proteins and lectins isolated therefrom
INVENTOR(S): Kim, Jongbae; Song, Seongkyu; Suh, Byungsun; Lee, Kwanee; Doo, Myoungsool; Kwak, Jinhwan; Song, Byeoungdoo; Yoon, Taekjoon; Kang, Taebong; Park, Choonho
PATENT ASSIGNEE(S): Mistle Biotech Co., Ltd., S. Korea
SOURCE: Eur. Pat. Appl., 62 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1074560	A2	20010207	EP 2000-402168	20000727
EP 1074560	A3	20030102		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
KR 2001011330	A	20010215	KR 1999-30638	19990727
US 6846913	B1	20050125	US 2000-627165	20000727
PRIORITY APPLN. INFO.:			KR 1999-30638 A	19990727

AB Disclosed is an extract from Korean mistletoe KM-110, which is of immunity enhancement and activity against tumor metastasis and can be used as an adjuvant material for vaccines applicable for the induction of humoral and cell-mediated immunity. Also disclosed are its fractions, a protein fraction KM-AS, a lectin fraction KML-C, lectin components KML-IIU and KML-IIL, which both are further purified from lectin fraction KML-C, a protein KMHBP which is obtained through heparin binding chromatog. eluting with NaCl from a fraction C-free AS which is a portion of the KM-AS free of KML-C, and a mixture KM of the KMHBP and the KML-C. They are revealed to their roles in the humoral and cell-mediated immunity enhancement and antitumoral activity.

IT 324745-98-4
RL: PRP (Properties)

09890219

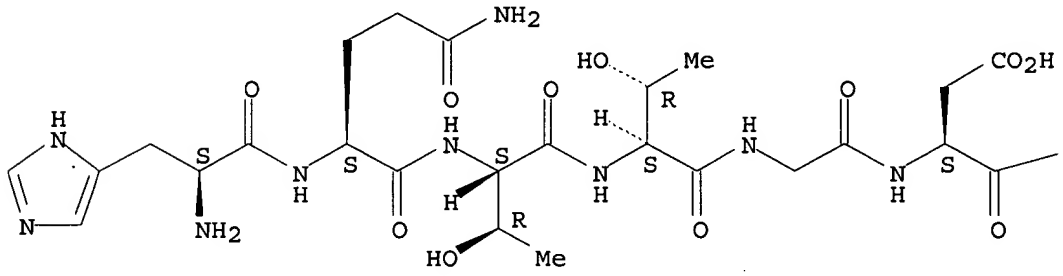
(unclaimed sequence; crude extract from Viscum album coloratum, and proteins and lectins isolated therefrom)

RN 324745-98-4 HCAPLUS

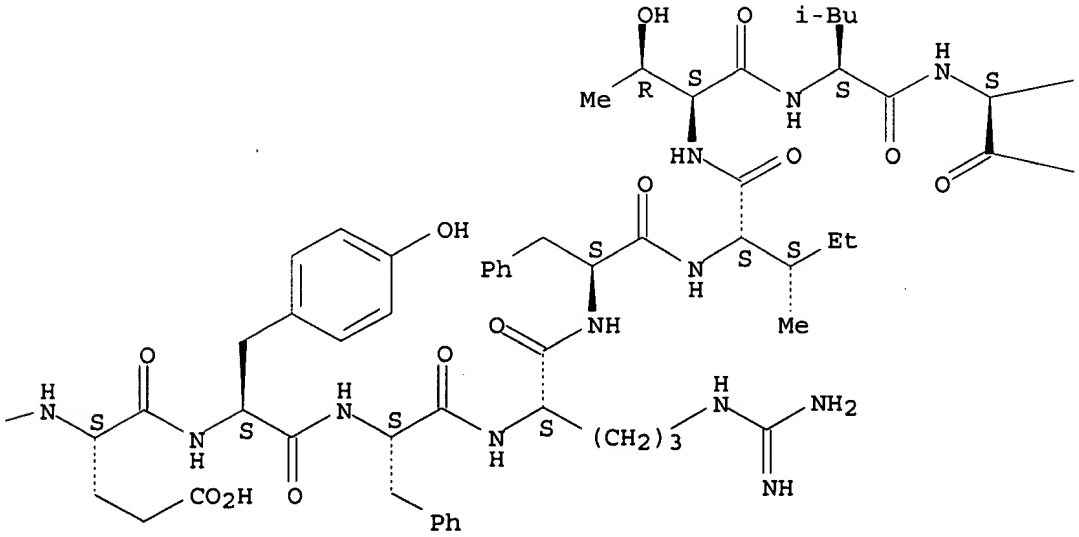
CN L-Aspartic acid, L-histidyl-L-glutaminyl-L-threonyl-L-threonylglycyl-L-α-aspartyl-L-α-glutamyl-L-tyrosyl-L-phenylalanyl-L-arginyl-L-phenylalanyl-L-isoleucyl-L-threonyl-L-leucyl-L-leucyl-L-arginyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

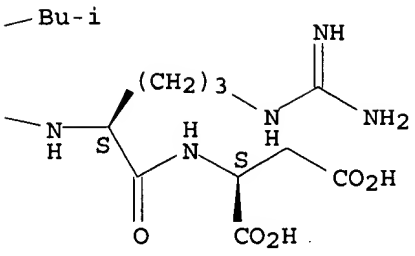
PAGE 1-A



PAGE 1-B



Updated Search



L15 ANSWER 5 OF 5 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1995:339375 HCAPLUS
DOCUMENT NUMBER: 123:9927
TITLE: Preparation of cis-epoxide peptide derivatives useful as irreversible HIV protease inhibitors.
INVENTOR(S): Kim, Sung Chun; Choy, Nakyen; Lee, Chang Sun; Son, Young Chan; Choi, Hoil; Koh, Jong Sung; Yoon, Heungsik; Park, Chi Hyo; Kim, Sang Soo
PATENT ASSIGNEE(S): Lucky Ltd., S. Korea
SOURCE: Eur. Pat. Appl., 95 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 601486	A1	19940615	EP 1993-119458	19931202
EP 601486	B1	19971029		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
KR 9600077	B1	19960103	KR 1992-23088	19921202
KR 9600078	B1	19960103	KR 1992-23089	19921202
KR 9711577	B1	19970712	KR 1993-10811	19930614
KR 9611580	B1	19960824	KR 1993-21299	19931014
KR 125104	B1	19971205	KR 1993-21298	19931014
KR 134497	B1	19980421	KR 1993-21300	19931014
JP 07002820	A	19950106	JP 1993-303063	19931202
JP 2916359	B2	19990705		
AT 159728	T	19971115	AT 1993-119458	19931202
ES 2111700	T3	19980316	ES 1993-119458	19931202
US 5744621	A	19980428	US 1996-667888	19960620
US 5763631	A	19980609	US 1996-667133	19960620
JP 10081653	A	19980331	JP 1997-214411	19970808
JP 2978848	B2	19991115		

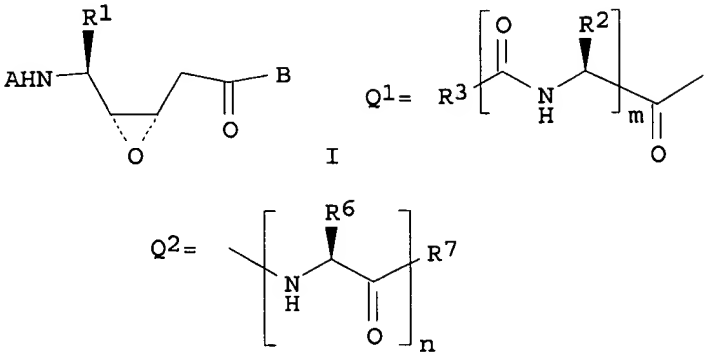
PRIORITY APPLN. INFO.:			
KR 1992-23088	A	19921202	
KR 1992-23089	A	19921202	
KR 1993-10811	A	19930614	
KR 1993-21298	A	19931014	
KR 1993-21299	A	19931014	
KR 1993-21300	A	19931014	

Updated Search

09890219

US 1993-159382 A3 19931130
JP 1993-303063 A3 19931202

OTHER SOURCE(S): MARPAT 123:9927
GI



I

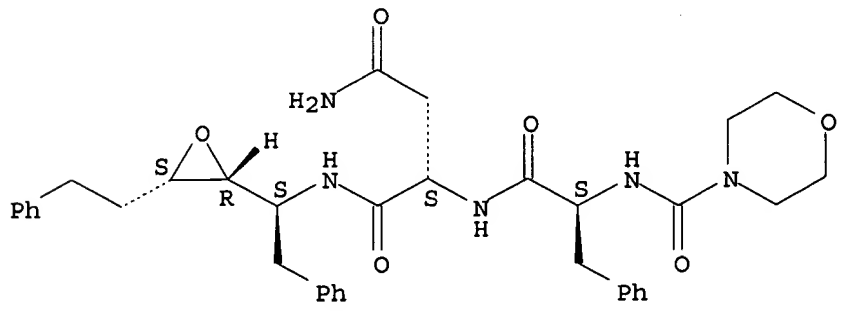
AB Title compds. [I; R1 = cycloalkyl, arylalkyl; A = Q1; R2 = (amide-substituted) alkyl; R3 = alkoxy, aryloxyalkyl, arylalkoxy, N-containing aromatic radical, N-containing aromatic radical-substituted alkoxy, amino; B = Q2;
R6 = alkyl, aralkyl, amide-substituted alkyl; R7 = alkoxy, alkylamino, alkoxyamino, dialkylamino, etc.; m = 0,1; n = 1,2], were prepared Thus, N-[5-L-(N-benzyloxycarbonylamino)-(4R,3S)-epoxy-6-phenylhexanoyl]isoleucine Me ester (preparation given) was hydrogenolyzed in MeOH over Pd/C; the residue was coupled with N-(2-quinolinecarbonyl)asparagine using EDC/HOBT/Et3N in DMF to give N-[5-L-[[N-(2-quinolinecarbonyl)asparaginyl]amino]epoxy-6-phenylhexanoyl]isoleucine Me ester. The latter inhibited HIV protease with KI = 0.018 μM, and inhibited HIV proliferation in cell culture with IC50 = 0.2 μM.

IT 160743-23-7P
RL: SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of, as HIV protease inhibitor)

RN 160743-23-7 HCAPLUS

CN L-Aspartamide, N-(4-morpholinylcarbonyl)-L-phenylalanyl-N1-[2-phenyl-1-[3-(2-phenylethyl)oxiranyl]ethyl]-, [2R-[2α(S*),3α]]- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



Updated Search

09890219

Updated Search